EMERGENCY WAR SURGERY

EMERGENCY WAR SURGERY

FIRST UNITED STATES REVISION OF

THE EMERGENCY WAR SURGERY NATO HANDBOOK

S.E.A. PUBLICATIONS

FULLERTON, CALIFORNIA

EMERGENCY WAR SURGERY

© 1982
Desert Publications

Reprinted under license from Desert Publications by:

S.E.A. PUBLICATIONS
2515 W. Orangethorpe
Fullerton, California 92633

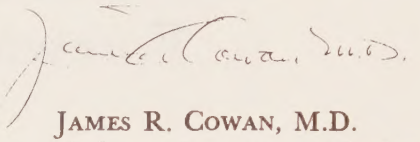
Foreword

A handbook on Emergency War Surgery was published originally by SHAPE (Supreme Headquarters Allied Powers Europe) in 1957. The NATO (North Atlantic Treaty Organization) nations agreed, by Standardization Agreement 2068, to standardize the principles of the initial care of war wounded as put forth in the SHAPE Handbook. The familiar Department of Defense hard-bound Emergency War Surgery NATO Handbook was published in 1958 as the United States implementation of the NATO agreement. A limited paperback edition was reprinted in 1959 without change except for the appendix on mouth-to-mouth resuscitation. Since publication of the first edition of the Handbook, advances in the treatment of war wounds, as well as in civilian surgery, were determining factors in the decision to revise the Handbook in its entirety. A tri-service editorial committee under the auspices of The Surgeon General, Department of the Army, was appointed to accomplish this task. The Editor in Chief of this volume was Brig. Gen. Thomas J. Whelan, Jr., MC USA (Ret.).

The Handbook incorporates extensive contributions from members of the SHAPE Revision Committee from France, the Federal Republic of Germany, the Netherlands, and the United Kingdom.

The editorial committee is indebted to many consultants, military and civilian, who submitted detailed recommendations on chapters dealing with their fields of special expertise. The committee is especially grateful for the advice and encouragement of Brig. Gen. Sam F. Seeley, MC USA (Ret.), who was chairman of the NATO committee that provided the original Emergency War Surgery Handbook. Particular credit is due to

the coeditors, Rear Adm. Edward J. Rupnik, MC, USN; Col. Robert M. Dean, USAF (MC); and Col. Richard R. Torp, MC, USA, who, with dedication and enthusiasm for the effort, helped to make this revision possible.



JAMES R. COWAN, M.D.
Assistant Secretary of Defense
(Health and Environment)

Preface

This revision of the Emergency War Surgery Handbook updates the care of the wounded necessitated by recent improvements and innovations in such care, arising from experiences in recent conflicts.

In general, the original format of the Handbook has been preserved, and many parts of the original text have required little or no change. Certain chapters and portions of chapters, however, have been deleted or replaced.

Sections dealing with mass casualties at the end of each chapter in the original text have been eliminated and replaced by a separate chapter, Mass Casualties in Thermonuclear Warfare; Radiation Injuries in the original Handbook has also been replaced by this new chapter. Original Chapter V, Crush Injury, has been deleted and its contents have been incorporated into an expanded chapter, the present Chapter XVI, Wounds and Injuries of the Soft Tissues.

In addition, two new chapters have been added: Aeromedical Evacuation and Reoperative Abdominal Surgery. These changes have required renumbering some of the old chapters, and the titles of a few of the old chapters have been changed slightly.

The number of illustrations has been increased from 18 to 34, and additional tabular data have been included in the text. The illustrations were completely redone by Mrs. Elisabeth W. McDonnell of the Scientific Illustration Division of the Armed Forces Institute of Pathology, to whom the editorial board owes a special debt of gratitude.

To those members of the Medical Corps of each of the Military Departments and to the civilian consultants who rewrote or reviewed the chapters, the editorial board is greatly indebted.

Sincere appreciation is extended to the members of the editorial board for the long hours of hard work which they unself-

ishly dedicated to the task. Personnel of The Historical Unit, U.S. Army Medical Department, were of inestimable assistance as coordinators of the entire project from its research phase through publications editing. Grateful appreciation is expressed to Mrs. Mary D. Nelson for publications editing of the Handbook and preparation of the index and to Mrs. Thelma L. Trout for organization of the manuscript and clerical support.

Finally, the editorial committee owes a special vote of thanks for the efforts and cooperation of Col. Tommy A. Pace, RAMC, as the Chairman, Emergency War Surgery Handbook Revision Committee, SHAPE, and his committee consisting of representatives from the United Kingdom, France, the Federal Republic of Germany, and the Netherlands, who approved this United States revision as a suitable model for the SHAPE revision.

Thomas J. Whelan, Jr.

THOMAS J. WHELAN, JR., M.D.
Editor in Chief
Brigadier General, USA (Ret.)

Contents

		Page
Foreword		iii
Preface		v
Chapter		
I	General Considerations of Forward Surgery	1
	Echelons of Medical Care	2
	Mass Casualties	4
	Part I. Types of Wounds and Injuries	
II	Missile-Caused Wounds	9
	Fundamentals of Wound Ballistics	9
	Distribution of Missile Wounds	16
	Clinical Considerations	17
III	Burn Injury	18
	Etiologic Agents	19
	Magnitude of Injury	19
	Depth of Injury	20
	Pathophysiology	22
	First Aid	22
	Initial Treatment of Extensive Burns	23
	Wound Care	30
	Electric Injury	31
	Chemical Burns and White Phosphorus Injury	31
	Topical Chemotherapy	33
	Evacuation	34
IV	Cold Injury	35
	Classification	35
	Pathogenesis and Pathologic Process	37
	Epidemiology	38
	Clinical Manifestations	41
	Management	43
	Prophylaxis	46

Chapter		*Page*
V	Blast Injury	48
	Types of Blast Injury	48
	Clinical Manifestations and Diagnosis	50
	Regional Injuries	50
	Preventive Measures	54
VI	Chemical Injury	55
	General Principles of Management	56
	Nerve Agents	57
	Vesicants (Blister Agents)	58
	Irritants (Tear and Nose Agents)	58
	Cyanogen Agents	58
	Lung Damaging Agents (Choking Agents)	59
	Incapacitants	60
	Other Agents	61
VII	Mass Casualties in Thermonuclear Warfare	62
	General	62
	Medical Effects of Nuclear Weapons	65
	Thermal Burns	65
	Blast Injuries	67
	Translational Injuries	69
	Radiation Injuries	69
VIII	Multiple Injuries	82
	Etiologic Considerations	82
	Management	83
	Part II. Response of the Body to Wounding	
IX	Shock and Resuscitation	91
	Pathogenesis and Etiologic Factors	91
	Predisposing and Aggravating Factors	92
	Clinical Manifestations	93
	Organization of a Triage and Resuscitation Facility	95
	Treatment	97
X	Physiologic Responses to Trauma	104
	Cardiovascular Response	104
	Hormonal Response	105
	Water and Electrolyte Responses	106
	Pathologic Responses to Trauma	108
XI	Infection	119
	General Considerations	119
	Etiologic Factors	120
	Bacteriology	121
	Classification of Wound Infection	123

Chapter *Page*

XI Infection—Continued
Antibiotic Therapy 126
Clostridial Myositis 133
Anaerobic Cellulitis 143
Streptococcal Myositis 144
Tetanus 144

Part III. General Considerations of Wound Management

XII Sorting of Casualties 153
General Considerations 153
Evacuation 154
Air Evacuation From the Battlefield 155
Sorting at the Battalion Aid Station 156
Sorting at the Level of Initial Wound Surgery 157
Concepts of Sorting 157
Priorities of Treatment 158
XIII Aeromedical Evacuation 159
Introduction 159
Aircraft 159
Special Considerations 160
XIV Primary Treatment in the Division Area 165
Care at the Battalion Aid Station 165
Maintenance of Cardiorespiratory Function 166
Control of Hemorrhage 177
Control of Shock 178
Dressings and Splints 179
Control of Infection 179
Relief of Pain 180
Hydration 180
Recording 180
Evacuation 181
Regional Injuries 181
Treatment in Brigade Medical Company or Division Clearing Station 185
XV Anesthesia and Analgesia 186
Anesthesia Equipment 187
Preoperative Preparation 188
Techniques of Anesthesia Induction 189
Postoperative Management 195
XVI Wounds and Injuries of the Soft Tissues 196
Principles of Management 196
Preoperative Preparation 197

Chapter		Page
XVI	Wounds and Injuries of the Soft Tissues—Continued	
	Technique of Debridement (Wound Excision)	198
	Crush Injury	203
	Management of Infected Wounds	206
	Postoperative Management and Evacuation	206
XVII	Vascular Injuries	208
	General Principles	209
	Diagnosis	209
	Surgical Timing	210
	Treatment	211
	Postoperative Management	220
	Adjunctive Therapy	222
	Complications	222
	Results	224
XVIII	Wounds and Injuries of Bones and Joints	225
	General Principles	225
	Management by Field Medical Personnel	226
	Management at the Forward Hospital	227
	Fractures	227
	Joint Injuries	231
	Redebridement and Wound Closure	232
	Infected Bone or Joint Injuries	232
	Sprains and Dislocations	233
XIX	Wounds and Injuries of Peripheral Nerves	234
	Classification of Injury	234
	Initial Wound Care	235
	Closed Nerve Injuries	236
	Splinting	236
	Causalgia	236
XX	Amputations	238
	Indications	239
	Technique	239
	Postoperative Management	243
	Stump Wound Closure	243
	Transportation	243
	General Principles	244

Part IV. Regional Wounds and Injuries

XXI	Craniocerebral Wounds and Injuries	247
	Classification	247
	Neurological Evaluation	248

Chapter		*Page*
XXI	Craniocerebral Wounds and Injuries—Continued	
	Management	249
	Expedient Measures	256
	Prognosis	256
XXII	Wounds and Injuries of the Spinal Cord	257
	Evaluation	258
	Management Principles	259
	Closed Wounds	259
	Open Wounds	260
	Reduction and Stabilization of Cervical Injuries	262
	Supportive and Postoperative Care	262
XXIII	Maxillofacial Wounds and Injuries	267
	Diagnosis	268
	Initial Management	268
	Initial Wound Surgery	270
	Fracture Management	271
	Postoperative Management	273
	Regional Fractures	273
	Evacuation	279
XXIV	Wounds and Injuries of the Eye	281
	Examination and Diagnosis	282
	Management	283
XXV	Wounds and Injuries of the Ear	291
	The External Ear	291
	The Middle Ear	293
	Aero-Otitis	293
	The Inner Ear	294
XXVI	Wounds and Injuries of the Neck	295
	Anesthetic Considerations	296
	Wounds of the Larynx and Trachea	296
	Wounds of the Pharynx and Esophagus	297
	Vascular Injuries	298
	Nerve Injuries	300
	Emergency Tracheostomy	300
XXVII	Wounds and Injuries of the Chest	303
	Pathophysiology	303
	Principles of Management	307
	Management in the Division Area	308
	Evacuation	310
	Management in the Forward Hospital	311
	Postoperative Management	314

Chapter *Page*

XXVIII Wounds and Injuries of the Abdomen ... 315
Preoperative Sorting ... 316
Operation ... 317
Postoperative Management ... 332
Postoperative Peritonitis ... 335
Evacuation ... 339
XXIX Reoperative Abdominal Surgery ... 340
Time of Reoperation ... 340
Specific Reasons for Reoperations ... 341
XXX Wounds and Injuries of the Genitourinary Tract ... 347
Wounds of the Kidney ... 347
Wounds of the Ureter ... 348
Wounds of the Bladder ... 348
Wounds of the Urethra ... 349
Wounds of the External Genitalia ... 350
XXXI Wounds and Injuries of the Hand ... 352
Care in the Division Area ... 353
Initial Wound Surgery ... 354
Wound Closure ... 356

APPENDIXES
A Glossary of Drugs With National Nomenclatures ... 358
B Useful Tables ... 364

INDEX ... 369

Illustrations

Figure

1 Temporary pulsating cavity produced by high-velocity missile ... 12
2 Energy transfer from missile to tissue ... 13
3 Yawing of bullet ... 15
4 Tumbling of bullet ... 15
5 Precession of bullet ... 15
6 Nutation of bullet ... 16
7 Rule of nines (distribution of body surface area) ... 20
8 Fallout decay with time after detonation ... 71
9 Typical blood cell response to whole blood irradiation ... 76
10 Insertion of subclavian catheter ... 99
11 Exhaled air methods of artificial respiration ... 170
12 Chest pressure-armlift method of Silvester ... 173
13 External cardiac compression ... 175

Figure *Page*
14 Technique of debridement in soft-tissue wounds 200
15 Repair of arterial injury 215
16 External fixation 229
17 Immobilization of upper extremity 230
18 Technique of open circular amputation 241
19 Self-contained traction device in plaster cast 242
20 Technique for debridement of head wounds 252
21 Locations of exploratory burr holes and incisions 255
22 Stryker frame improvised from standard litters 261
23 Technique of application of Crutchfield tongs 263
24 Technique of immobilization of injured jaw 272
25 Exposure of subclavian vessels 299
26 Technique of tracheostomy 301
27 Effect of open chest wound on respiration 305
28 Control of bleeding from caval and hepatic vein injuries 321
29 Drainage of abdomen 323
30 Suture technique for small bowel injuries and anastomoses 326
31 Exteriorization of colon wounds 327
32 Matured ileostomy and end colostomy 328
33 Presacral drainage through posterior incision 330
34 Immobilization of injured hand and forearm 353

Tables

Number
1 Missile injuries in World War II, Korean war, and Vietnam war. 17
2 Diagnosis of depth of burns 21
3 Brooke formula for fluid requirements in burn patients 24
4 Suggested choices of antimicrobial agents 128
5 Dosage and administration routes of antimicrobial agents 134
6 Anesthetic agents 190
7 Arterial wounds and associated injuries, Vietnam, 1965–70 221

CHAPTER I

General Considerations of Forward Surgery

Military surgery is a development within the art and science of surgery which is designed to carry out a specialized, essential, and highly significant mission under the adverse conditions of war

The additional necessity of haste in caring for a continuous flow of battle casualties does not mean that military surgery is carried out in an atmosphere of confusion and disorder or that standard principles of treatment are abandoned. On the contrary, as all past military medical history shows, intelligent planning and training, in anticipation of the needs of the emergency, have made possible an enviable record in military medicine.

The basis of success in military medicine in the combat zone is an organized team, each member of which has been trained to accept the responsibilities of his assigned position and to be prepared to move to a new station, with different responsibilities, as new situations develop. The system for the evacuation and management of casualties is such as to demand that most patients will be treated by several surgeons, in team and in succession, and not by a single practitioner. No matter how expert a medical officer may be in one field or another, he must always conduct himself within the purposes and limitations of the mission of the particular medical echelon in which he finds himself at the moment.

Success in military medicine, furthermore, has been achieved

despite the fact that, over the ages, many—sometimes most—of the lessons of the past, all learned by hard experience, ordinarily lie fallow between conflicts. Almost invariably, they have had to be rediscovered, relearned by additional hard experience, and expanded and adapted by succeeding medical generations as new emergencies have arisen.

The milestones of history, unfortunately, very often are represented by wars, and modern wars may not be limited conflicts between nations. Instead, they may be fought between groups of nations. The role of the medical profession, therefore, extends to the care of collaborating nationalities. It must be carried out in widespread geographic areas and in extremes of climate. These facts, highlighted by the continuing tensions of the times in which we live, explain the need for the expansion of the curriculums of medical education to include the doctrines and principles of military medicine. Some medical students will make military medicine their careers. Those who do not may be called upon in emergency to serve in the medical department of some branch of the Armed Forces. They too must know the fundamentals of military medicine. Military medical knowledge, in short, is no longer a function of professional medical officers alone.

ECHELONS OF MEDICAL CARE

A basic characteristic of the organization of modern military medical services is the distribution of medical resources and medical capability in facilities at various stages or levels of location and function which are referred to in formal military parlance as "echelons." Echelonment is a matter of principle, practice, and organization pattern, not a matter of rigid prescription. Scope of function may be expanded or contracted on sound indication; one or more echelons may be bypassed on grounds of efficiency or expediency; and formal organizational structure will differ with time and among various armed forces. The following general pattern, however, is usually apparent.

Military medical care is carried out in the following echelons:

1. In the *first echelon,* the trained medical aidman provides first aid and conveys or directs the casualty to the battalion aid

station. Because of the proximity of the aid station to the battleline, its mission is simply to provide essential emergency care and to prepare the casualty for evacuation to the rear. This care may include the beginning of intravenous fluid administration, the control of hemorrhage, and the establishment of an airway.

2. In the *second echelon,* care is rendered at an assembly point or clearing station. Here the casualty is examined, and his wounds and general status are evaluated, to determine his priority, as a single casualty among other casualties, for continued evacuation to the rear. Emergency care, including beginning resuscitation, is continued, and, if necessary, additional emergency measures are instituted; but they do not go beyond the measures dictated by the immediate necessities. This function is performed typically by company-size medical units organic to brigade or division.

3. In the *third echelon* of care, the casualty is treated in a medical installation staffed and equipped to provide resuscitation, initial wound surgery, and postoperative treatment. Casualties whose wounds make them nontransportable receive surgical care in a hospital close to the clearing station. Those whose injuries permit additional transportation without detriment receive surgical care in a hospital farther to the rear.

4. In the *fourth echelon* of medical care, the casualty is treated in a general hospital staffed and equipped for definitive care. General hospitals are located in the communications zone, which is the support area to the combat zone or army area. The mission of these hospitals is the rehabilitation of casualties to duty status or, if rehabilitation cannot be accomplished within the permitted holding period, their evacuation to the Zone of Interior for reconstructive surgery and other treatment.

Patients with wounds of lesser severity may not need to be passed through all echelons. When aeromedical evacuation is readily available, the first, second, or, rarely, third echelon of medical service may be bypassed. Specific surgical functions of the first three echelons are described in greater detail in subsequent chapters.

It is important to remember that there is a logistic problem in the care of all battle casualties. Military medical facilities

always must be in a state of readiness to move according to the dictates of the tactical situation, though this necessity in no way lessens the responsibility of the medical service for providing for the medical care and disposition of casualties.

Despite the exceedingly unfavorable circumstances of war, movement of casualties from echelon to echelon in the forward area usually is accomplished within a matter of hours. Distances vary with the local tactical situation, but, generally speaking, casualties travel a distance of many miles between the battlefront and a hospital.

Because the individual who has been wounded in combat is cared for by multiple surgeons at different echelons of medical care and because hospitals may be separated by great distances, the consultant system has been developed. Certain individuals selected as consultants because of their expertise in a given specialty field have been utilized to correlate end results as noted in hospitals of the communications zone with initial surgical care in the combat zone. The responsibility of feedback to the forward surgeon and evaluation of the effectiveness of combat surgery reside with these individuals. In addition, professional meetings of practicing surgeons from both combat and communication zones have been utilized to exchange views on methods of surgical care. For example, annual War Surgery Conferences were held to bring American surgeons at all levels and in all branches of the armed services up to date on the latest information and results in the care of the wounded in the Vietnam war.

Modern concepts of increased mobility for all fighting units, as well as the vulnerability of even remote areas to aerial or missile attacks, require that all medical units, wherever they are located and whatever their original mission, must be prepared to receive and treat casualties as circumstances require. In modern warfare, the battlefront is likely to be highly fluid. Medical officers must be prepared to adjust themselves realistically to urgent needs as they arise.

MASS CASUALTIES

A separate chapter in this handbook is devoted to thermonuclear warfare and the management of mass casualties. The

term "mass casualties" denotes the sudden inundation by an overwhelming number of casualties within a brief period of time, with locally available medical facilities completely unable to supply ordinary medical care for them. The methods suggested, which frankly are based on compromises, are applicable only to mass casualties. They are not intended to apply to a situation which is more common in conventional warfare; namely, the temporary overtaxing of local medical facilities by a casualty load of unusual proportions. It is assumed, furthermore, that, as soon as the disparity between mass casualties and limited medical capabilities has been overcome, conventional principles and practices again will be adhered to, just as in the more usual circumstances of military surgery.

The application of thermonuclear energy to instruments of war, together with the proved capabilities of this new force for mass destruction, has brought about far-reaching changes in all military planning and training to cope with the flood of problems introduced by these new developments. In thermonuclear warfare, the basic principle of military medicine remains unchanged: the salvage of the greatest possible number of lives for the support of the military mission. This objective can best be accomplished by (1) preliminary appreciation of the necessities of the emergency and (2) preplanned adjustments in the optimal conventional standards of the care of casualties.

PART I

Types of Wounds and Injuries

CHAPTER II

Missile-Caused Wounds

Certain fundamental knowledge concerning wound ballistics is essential to the military surgeon. The term "wound ballistics" is defined as the study of the motion of missiles within the body and the wounding capacity of various missiles. Identification of causative agents of wounds is important for two reasons—medical and tactical. From the medical standpoint, the surgeon must know the wounding capacity of the weapon to assist in evaluating the extent of injury to achieve the most effective therapy. From the tactical point of view, the effectiveness of special weapons can be gaged correctly by careful and astute observation of the nature of the wounds produced by these weapons.

FUNDAMENTALS OF WOUND BALLISTICS

The wounding potential of any missile is the summation of numerous factors which together constitute the principles of wound ballistics. The behavior of the missile in relation to wounding capacity includes the following:

1. The amount of energy transmitted to the body upon impact by a missile is determined in part by the mass and size of the missile, but chiefly by its velocity.

2. The amount of energy expended is expressed by the formula:

$$KE = \frac{m(v_1^2 - v_2^2)}{2g}$$

where KE refers to kinetic energy, m refers to mass, v_1 stands for the initial velocity, v_2 stands for the final velocity, and g refers to acceleration due to gravity. As this formula shows, velocity is the most important factor in the production of the kinetic energy expended in the body upon wounding.

3. The behavior of the missile upon penetration is influenced by the density of the tissues injured and the elasticity of the skin and other tissues.

4. The direction of the transmitted energy is determined by the shape of the bullet, its motion in flight, and stability at the time of impact.

Mass and Size of the Missile

The number of high-velocity bullet wounds has been increasing in succeeding wars of modern times; the increase resulted from the use of high-velocity small arms and automatic weapons together with reintroduction of the Claymore mine which, upon detonation, emits numerous spherical missiles at high velocity. The AK–47 Russian military rifle utilized bullets weighing 122 grains as compared to the U.S. high-velocity M–16 rifle bullet weighing 55 grains. Artillery, mortar, and mine fragments are irregular and vary not only in size and shape but also in velocity. Both bullets and fragments are considered to be primary missiles. Secondary missiles include shell wadding, clothing, building material, rocks, and other objects which are converted into wounding missiles by the effects of the primary missile.

Velocity

Velocity, the most important factor in wounding, is expressed in feet per second or in meters per second. The terms "high velocity" and "low velocity" are arbitrary. Any missile traveling more than 2,000 feet per second can be considered a high-velocity missile, although often 2,500 feet per second is chosen as the dividing line. Civilian injuries usually are inflicted with low-velocity weapons. The .38 caliber pistol, for example, has

a muzzle velocity of approximately 680 feet per second. This weapon can be compared to the high-velocity military rifle bullet (M–16 bullet) which has a muzzle velocity of approximately 3,250 feet per second. The kinetic energy theory of wounding capacity indicates that the energy expended is directly proportional to the mass and to the square of the effective velocity. Thus, doubling of the velocity quadruples the kinetic energy of the missile. Rifles employed in World War I had an average velocity of 2,300 feet per second (700 meters per second), while those employed in the Vietnam conflict developed a muzzle velocity in excess of 3,200 feet per second (976 meters per second).

Low-velocity missiles.—The mechanism by which a low-velocity missile produces a wound is relatively simple. In a knife or bayonet wound, for example, only those tissues with which the blade comes into contact are severed. No significant energy is imparted to the nearby tissues, and the damage, for all practical purposes, is entirely localized. The low-velocity bullet or fragment also creates local damage and has been shown to push the tissue aside. In all wounds produced by low-velocity missiles, therefore, only minimal debridement is required. The impact of high-velocity missiles which become spent also produces wounds equivalent to those produced by low-velocity missiles.

High-velocity missiles.—The most important point to be learned in the transition of the civilian physician to the military surgeon is that the high-velocity wound is a totally different entity. The kinetic energy of the missile upon impact becomes imparted to the tissues. These adjacent tissues are suddenly hurled forward, laterally, and then backward. This is the theory of the acceleration of particles. A large space or cavity is created approximately 30 to 40 times the size of the missile, in which pressures of 100 atmospheres (1,500 psi) have been recorded (fig. 1). This effect was first termed "cavitation" by Woodruff in 1898. The temporary cavity becomes a vacuum which draws in air from both the entrance and the exit sites (fig. 2). Its maximal size is attained within a few milliseconds and it pulsates quickly to rest, forming the permanent cavity. This permanent wound tract is visible to the clinician and the

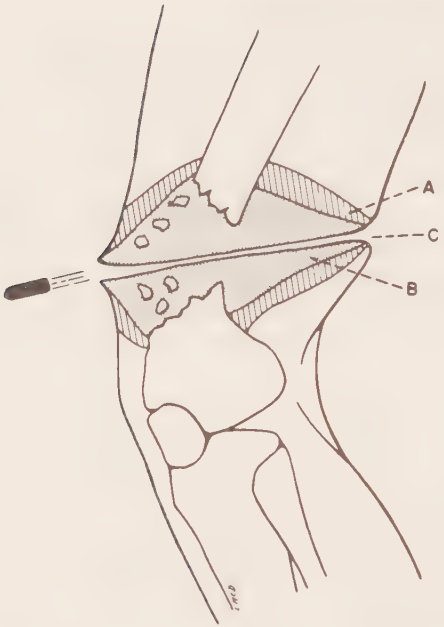


FIGURE 1.—Temporary pulsating cavity produced in an extremity perforated by a high-velocity missile. A. Full extent of the area of cavitation produced by transfer of energy from the missile to the tissue which is violently flung away from the missile tract. B. The receding wave of compressed tissue smaller in size than the original area of cavitation. C. The residual missile tract which will remain as a visible tissue defect after all energy has been expended and the tissue has come to rest.

damage at a distance may not be immediately apparent. The effect of this cavity becomes particularly more damaging when the bone is fragmented and acts as a secondary missile. Experimentation has shown that the high-velocity missile neatly shears the tissue while the violence of the expansion of the missile tract, as the cavity is created, disrupts tissues, ruptures blood vessels and nerves, and may even fracture bones at distances removed from the path of the missile. This force of the cavity simulates compression or crushing injuries similar to those sustained in blunt trauma.

The explosive nature of high-velocity wounds was first observed by Huguier in 1848. Wilson stated in 1921 that a high-

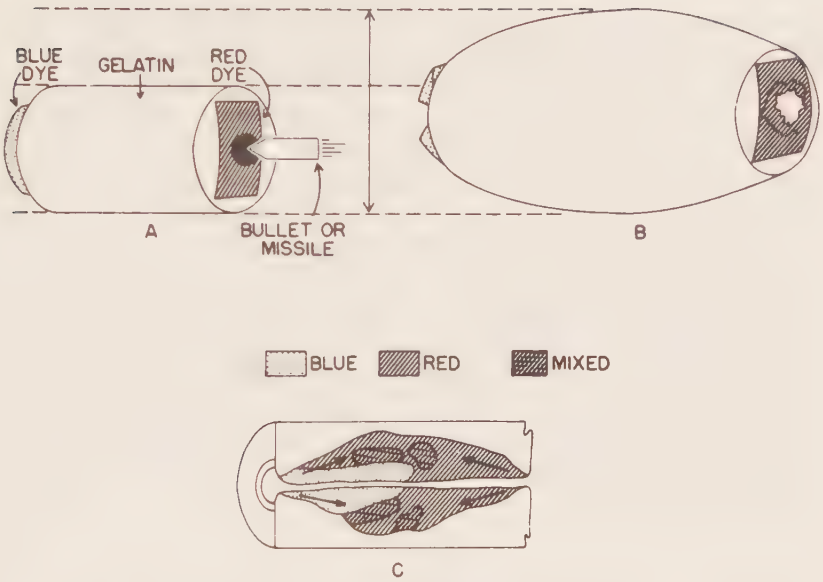


FIGURE 2.—Energy transfer from missile to tissue results in alternating positive and negative waves of pressure accompanying tissue cavitation. A. Cylinder of gelatin with a packet of powdered red and blue dye at each end. B. Cylinder of gelatin at moment of impact (and cavitation) produced by a missile at 2,800 feet/second. C. Because of alternating waves of positive and negative pressure within the cavitation zone, red dye is carried in by the missile and blue dye is sucked in by negative pressure. This provides an explanation for the method of tissue contamination caused by perforating gunshot wounds.

velocity rifle bullet passing near a blood vessel produces intimal lesions at a distance from the missile tract. This concept was reinforced during the Korean war when vessels, the external appearance of which was grossly normal, were actually thrombosed. Recent experimentation has demonstrated that the force of kinetic energy may literally destroy a blood vessel when indirectly struck.

Tissue Destruction

The type of wound which results from a missile may be a simple contusion or a penetrating or perforating wound. In a

penetrating wound, the kinetic energy is dissipated wholly into the tissues. In a perforating wound, the amount of energy imparted to the tissues is the difference between the kinetic energy remaining at the point of exit and that present at the wound of entrance. The density of the tissues struck determines the nature of the wounds as described above. Kocher demonstrated in 1876 that tissues which contained large quantities of water were most severely damaged, and Daniel, in 1944, correlated the severity of high-velocity wounds with the specific gravity of the tissue involved. Muscle is severely damaged because of its relatively high homogenous density. In contrast, the lung sustains less extensive local destruction because of its low density, resulting in absorption of less energy and a smaller temporary cavity. Tissues of varying density, such as fascia or bone, may divert the direction of the missile, resulting in bizarre wound tracts.

Ballistic Behavior of Missiles

An understanding of the ballistic behavior of a missile is necessary to interpret accurately the mechanism of tissue destruction. Aerodynamic forces act upon the spin-stabilized bullet during flight and may change the angle of obliquity on impact. The effects of such forces include yaw, tumbling, precession, and nutation. Yawing is the deviation of a bullet in its longitudinal axis from the straight line of flight (fig. 3). Tumbling is the action of forward rotation around the center of mass (fig. 4). A bullet will tumble if its overturning moment due to yaw is sufficiently large. Spin, which maintains the bullet in a point on position, may introduce two circular motions: (1) precession, which is a circular yawing around the center of mass in a spiral fashion (fig. 5), and (2) nutation, which is the rotational movement in small circles forward in a rosette pattern (fig. 6).

The varying severity of wounds and amount of tissue destruction by identical missiles with the same energy can be explained, in most part, by the angle of the bullet to the tissues at the moment of impact. Thus, a nontumbling bullet entering the tissues at a right angle will be in minimal contact with the

FIGURE 3.—Yawing is the deviation of a bullet in its longitudinal axis from the straight line of flight.

FIGURE 4.—Tumbling is the action of forward rotation around the center of mass.

FIGURE 5.—Precession is a circular yawing around the center of mass in a spiral fashion.

tissues; upon exit, the bullet will have imparted a lesser amount of its kinetic energy to the tissues. A bullet which strikes at an oblique angle and tumbles through the tissues, on the other hand, will have contacted more tissue and consequently will have imparted a greater amount of its energy to the tissue, resulting in greater tissue destruction.

External appearances of both exit and entrance wounds may be misleading. The wound of exit is usually larger than the wound of entrance. The alert military surgeon will realize that, though the wounds caused by large fragments may appear to be more formidable, the energy imparted by smaller missiles of higher velocity may actually create far more serious and lethal injuries.

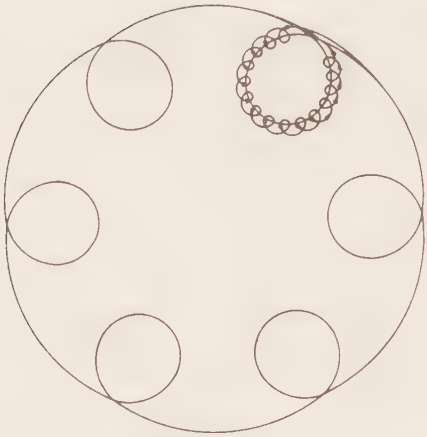


FIGURE 6.—Nutation is the rotational movement in small circles forward in a rosette pattern.

DISTRIBUTION OF MISSILE WOUNDS

In assessment of wound tracts and the distribution of wounds, one must ascertain, as accurately as possible, the position of the soldier at the time of wounding. When he is prone, for instance, a projectile which enters the shoulder may, by devious routes, penetrate the thorax or the abdomen and exit from the buttock area.

Although a soldier spends about two-thirds of his combat time in the prone firing position, recent combat experience showed that about 50 percent of individuals were wounded in the upright position, about 20 percent were wounded in a lying position, and smaller percentages were wounded while kneeling or sitting.

An analysis of wounds received over a 12-month period in Vietnam, compared to the wounds of World War II and Korea, is shown in table 1.

The distribution of these wounds may indicate the practical importance of protective measures in reducing the lethality of war wounds. The wearing of a steel helmet, for instance, reduces the severity as well as the incidence of head injuries. The use of an armored vest reduces the number of wounds of

TABLE 1.—*Distribution of missile injuries in World War II, Korean war, and Vietnam war*

Location	World War II	Korean war	Vietnam war[1]
	Percent	*Percent*	*Percent*
Head and neck	17	17	14
Thorax	7	7	7
Abdomen	8	7	5
Upper extremity	25	30	18
Lower extremity	40	37	36
Others	3	2	[2]20
Total	100	100	100

[1] Received over a 12-month period.
[2] Includes many multiple wounds in which there was no single predominant location.

the thorax and the abdomen. These protective devices will increase the proportion of nonfatal wounds, resulting in a greater survival rate.

CLINICAL CONSIDERATIONS

Awareness of the devastating effects of high-velocity wounds in combat has led to the firm surgical principle of wider and more complete debridement of all wounds. Such a debridement includes the removal of all devitalized tissue and foreign bodies. Those tissues which do not bleed promptly upon incision are regarded as devitalized. Structures destroyed at points distant to the actual missile tract favor the retention of devitalized tissues. The risk of infection in high-velocity wounds is increased because of the kinetic cavitation. The positive and negative cavitation effects introduce foreign materials and bacteria at both entrance and exit sites. Some observers believe, fallaciously, that bullets are sterile because of the heat generated in the firing process. Investigators as early as 1892 (LaGarde), however, described experiments in which bacterial infections were transmitted to animals shot with contaminated bullets. Thorsby, in 1967, demonstrated that heat-labile bacteria, *Serratia marcescens*, inoculated on missiles fired at 1,600 feet per second, uniformly grew along the bullet track. These facts should be well appreciated when planning the extent of wound excision.

CHAPTER III

Burn Injury

Extensive use of the various fuels needed to provide both ground and air mobility for the present-day Armed Forces has resulted in a large increase in the number of thermal burns encountered in military personnel. During times of conflict, the possibility of unintended ignition of these fuels is greatly increased, as is the chance of thermal injury from antipersonnel and other weapons. The development of thermonuclear devices has created the possibility of virtually instantaneous generation of large numbers of burn patients, creating not only medical but also severe logistical problems. Even under the best treatment conditions, the simultaneous receipt of multiple extensively burned patients at any hospital disrupts the activities of the professional and paramedical staff and places heavy demands upon the logistical system of that institution.

Recent laboratory developments and the clinically demonstrated efficacy of topical chemotherapy have resulted in general acceptance of simplified treatment techniques readily adaptable to the combat surgery environment.

In the management of the burn patient, first priority is given to maintenance of an airway, control of hemorrhage, and prompt institution of resuscitative therapy. The presence of associated traumatic wounds in the patient with burn injury may complicate the management of the burns and vice versa. The essence of successful treatment of burn patients with or without other traumatic injuries is effective triage, timely diagnosis, accurate assessment of surgical priority, and appropriate resuscitation.

ETIOLOGIC AGENTS

Ignition of gasoline and other fuels accounts for the greatest number of thermal injuries in the adult. Flame or flash burns may be caused by various other agents contained in explosives of particular importance in the combat zone. Patients with chemical burns and burns from phosphorus require immediate wound care in contrast to those with "conventional" burns (see p. 31). Thermal injury created by electric current also deserves separate consideration because of special treatment requirements.

Even in the combat zone, burns resulting from carelessness outnumber those resulting from hostile action. The enforcement of safety procedures and existing regulations will minimize such occurrences. The use of gloves, goggles, protective headgear, and flame-retardant clothing in high-risk personnel will also minimize, if not prevent, thermal injury in those individuals. This equipment is particularly important to fire-rescue personnel, and the use of these items should be enforced strictly, even (within limits) at the expense of personal comfort.

MAGNITUDE OF INJURY

The magnitude of thermal injury is dependent upon the depth and extent of the burn. These two factors determine not only mortality and initial treatment requirements but also morbidity, metabolic consequences of injury, character of healing, and the ultimate functional result.

The extent of the body surface burned can be estimated by employing the "rule of nines." The distribution of surface area by anatomical part in the adult is illustrated in figure 7, where the percent of skin surface is assigned to body part as follows: head and neck, 9 percent; anterior trunk, 18 percent; posterior trunk, 18 percent; upper extremities, 9 percent each; lower extremities, 18 percent each; and genitalia and perineum, 1 percent.

Burns of more than 15 percent of the body surface require some resuscitative treatment and, in most situations, necessitate hospitalization. The young adult (comprising the bulk of a

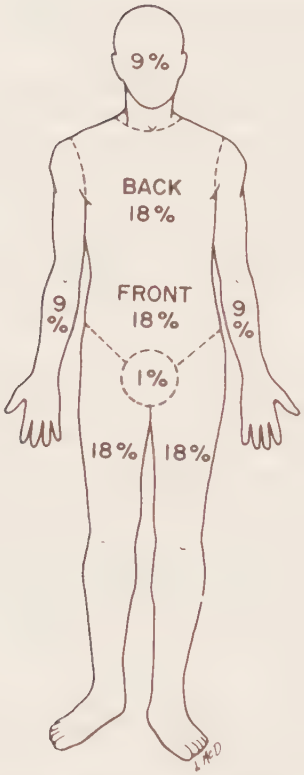


FIGURE 7.—Rule of nines, showing distribution of body surface area by anatomical part.

military population) tolerates thermal injury best, while older patients (above 50) and those in the very young age groups have a greater mortality rate for a given extent of burn. The location of the burn influences not only prognosis but also the need for hospitalization. Burns of the face, hands, feet, or perineum of small extent may require hospitalization, even if these limited areas are the only sites of burn injury.

DEPTH OF INJURY

The depth of thermal injury can be determined with certainty only by histologic examination. However, the clinical criteria in table 2 will permit an initial, usually quite accurate,

TABLE 2.—*Diagnosis of depth of burns*

Criteria	Second-degree burns	Third-degree burns
Cause	Hot liquids, flashes of flame.	Flame, electricity, chemicals.
Color	Pink or mottled red.	Dark brown or black; charred; translucent with thrombosed superficial veins visible; pearly white ±.
Surface	Vesicles or weeping.	Dry and inelastic.
Pinprick	Painful	Anesthetic.

differentiation between second- and third-degree burns. The total percentage of skin surface involved with second-degree and third-degree burns is the primary concern during resuscitation. Differentiation between second-degree and third-degree burns is more important later in the postburn course in assessing the metabolic demands, anticipated functional result, and ultimate need for autograft closure of the burn wound.

Those areas of thermal injury that are waxy-white, soft, and pliable, yet nonpainful, formerly were regarded as full-thickness injury, but in actuality are deep, partial-thickness burns; they frequently heal without the need for grafting if protected from invasive sepsis by topical chemotherapy. Charring with thermal injury of subcutaneous and deeper tissues is infrequent, but may occur in epileptics who are burned in the postictal state, patients trapped by burning debris or in a burning vehicle, or patients with significant high-voltage electric injury. Injuries of less than partial thickness—that is, first-degree burns (erythema of intact epidermis)—are important only so far as patient comfort and vasomotor lability are concerned and are, with few exceptions, treated symptomatically without need for resuscitation.

The depth of thermal injury after a thermonuclear explosion is dependent upon the intensity and duration of the thermal pulse, but burns also may be sustained from ignition of clothing or burning debris. These burns do not differ from burns of other etiology except for the associated effects of ionizing radiation, which decrease survival for a given-size burn.

PATHOPHYSIOLOGY

Thermal injury, regardless of the etiologic agent, results in cell death by coagulation necrosis. In areas of cell death and cell damage, capillary permeability is increased with loss of integrity of the vascular system and escape of the nonformed blood elements. This is manifested clinically by edema which forms most rapidly in the immediate postburn periods and reaches a maximum in the second postburn day. Thereafter, as vascular integrity is restored and fluid resorption begins, edema slowly resolves. This increase in capillary permeability results in a decrease in blood volume and an increase in blood viscosity, causing an increase in peripheral resistance and a decrease in cardiac output. Fluid resuscitation is carried out in the immediate postburn period to minimize these changes by maintaining blood volume at a level adequate for organ perfusion.

FIRST AID

The first consideration is removal of the source of thermal injury from the patient. Burning clothing should be extinguished and removed from the patient and the patient should be removed from burning vehicle or building. In electric injury, the patient should be removed from the point or points of contact, the rescuing personnel carefully avoiding contact with the power source. Chemical agents should be washed immediately from the skin surface by copious water lavage. First aid must be reduced to a minimum, and nothing must be done that could prejudice subsequent treatment. All constricting articles, such as rings, bracelets, wristwatches, belts, and boots, are removed, but the patient is not undressed. Makeshift wrappings, such as sheets, can be used, if necessary, to cover the burned areas, but special wrappings such as prepared burn dressings are better in protecting the injuries from contamination.

Patency of the airway should be assured, hemorrhage controlled, and fractures splinted. An intravenous pathway should be established in an unburned area if at all possible, and in an

upper extremity vein if there are associated abdominal wounds. Resuscitation may be safely begun with electrolyte solution alone and should be continued before and during movement to an installation where definitive medical care is available. An intravenous cannula is preferable in all situations since large volumes of fluid are required for patients with extensive burns, and patient restlessness, transportation, or edema may dislodge an intravenous needle. Patients with injuries from white phosphorus should have the burns dressed with saline-soaked dressings to prevent ignition of the phosphorus by contact with the air.

Pain is seldom a major problem in the patient with severe burns, but the patient with extensive partial-thickness burns may have considerable discomfort which can be relieved by appropriate doses of morphine or Demerol administered intravenously. Subcutaneous or intramuscular injections of analgesics will not be mobilized during the period of edema formation and will be ineffective in pain control. A patient who has received multiple subcutaneous or intramuscular doses of an analgesic may mobilize these simultaneously during the diuretic phase and develop severe respiratory depression which must be treated promptly.

On the day of injury, after hemorrhage is controlled, ventilatory stability achieved, and urinary output established, one should promptly move the extensively burned patient to a definitive treatment facility. Intravenous fluid administration should be maintained throughout transportation and, if any question exists as to adequacy of the airway, a tracheostomy should be performed.

INITIAL TREATMENT OF EXTENSIVE BURNS

At the definitive treatment facility, control of hemorrhage and airway adequacy again must be insured. Initial consideration of the burn patient includes a complete physical examination following removal of all the patient's clothing. Once a secure intravenous pathway has been established, one then must estimate and plan the resuscitation fluids to be given to the burned patient.

TABLE 3.—*Brooke formula for estimating fluid requirements in burn patients*

Fluid requirement	Type and amount
First 24 hours postburn:	
Colloid	Plasmanate, plasma albumin,[1] dextran[2]; 0.5 ml/% burn[3]/kg body weight.
Electrolyte solution	Lactated Ringer's, saline; 1.5 ml/% burn[3]/kg body weight.
5% Dextrose in water	2,000 ml added for evaporative water loss.
Second 24 hours postburn:	
Colloid	One-half to three-quarters of above.
Electrolyte solution	One-half to three-quarters of above.
5% Dextrose in water	2,000 ml.

[1] See text for details of proper use.
[2] Not to exceed 10 ml/kg or 1,000 ml total.
[3] Use no more than 50% for calculation.

Several formulas exist for calculation of the fluid needs of the burned patient on the basis of body weight and extent of burn. The Brooke formula, which has been employed successfully in several thousand burn patients, is detailed in table 3. Like any other formula, it should be modified according to the individual patient's response in terms of urine output, vital signs, and general condition. The extensively burned patient requires frequent examination and observation. Only in such a manner can his treatment be properly supervised and altered.

The initial 24-hour fluid requirements estimated by the formula are administered so that the patient receives half within the first 8 hours following injury, the time of most rapid edema formation. If the patient is received other than immediately following burn injury, the time interval which has elapsed must be subtracted from 8 hours and the first half of the resuscitation fluids administered in that period. A quarter of the estimated fluids then should be administered, ideally at a uniform rate, in each of the two succeeding 8-hour periods in the first 24 hours postburn.

The electrolyte-containing solution should be Ringer's lactate, which contains a more physiologic concentration of chloride, but isotonic saline may be employed if the former is not avail-

able. Even though red blood cell destruction occurs after thermal injury, whole blood is not administered as a portion of the resuscitation fluids. The increased capillary permeability, which would only lead to loss of the plasma and retention of the red cells, would further elevate the patient's hematocrit, adversely affecting the rheological properties of the blood. The colloid solution readily available within the military supply system is Plasmanate, a heat-treated plasma, free of hepatitis risk. Fresh plasma and albumin can be utilized if Plasmanate is not available, with appropriate fluid adjustments if albumin is employed (each gram bottle of albumin should be added to 500 cm^3 of normal saline for administration).

For purposes of calculation, in burns of over 50 percent, a maximum of 50 percent of the body surface is used for the burn size factor, although patients with these extensive burns usually will require more than the calculated fluid volumes. In general, one attempts to carry out resuscitation with the minimum fluid volume in excess of estimate necessary to maintain organ function and avoid pulmonary edema. In the larger burn, fluids are added to that amount calculated using a 50-percent maximum burn size, in the same proportion as calculated by the Brooke formula, with the additional fluid given as dictated by the patient's response. During the second postburn day, capillary integrity appears to be restored and resorption of edema fluid begins. Consequently, fluid needs diminish and the volumes required are usually between one-half to three-quarters of the first day's requirements of electrolyte and colloid-containing solutions. The adult patient again will require 2,000 ml of 5-percent dextrose in water to meet metabolic needs and evaporative losses from the burn surface. The relative distribution of total body surface area and fluid requirements in burns of children vary from those in adults.

Potassium supplements are not needed and may be deleterious during the first 48 hours since the serum potassium is commonly elevated because of destruction of red blood cells and other tissue. Therefore, potassium is lost from injured cells at a time when renal function may be depressed. From the third postburn day onward, potassium supplements should be added to the intravenous fluids if renal function is unimpaired.

Average daily potassium requirements range from 60 to 200 meq per day.

After the third postburn day, the burn patient usually will have a normal and, in some instances, a supranormal plasma volume, so that further administration of salt or colloid-containing fluids is usually unnecessary and should be carried out with great caution. The burn wound, following the third postburn day until healing or grafting, acts essentially as a free water surface with considerable evaporative losses; that is, 6 to 8 liters per day in a patient with a very extensive burn. The evaporative loss should be replaced as electrolyte-free water. Adequacy of hydration can be determined by careful monitoring of patient weight, serum osmolarity, and serum sodium concentration. Later in the postburn course, whole blood should be administered to maintain the hematocrit between 30 and 35 percent.

Urinary Output

The most readily available clinical guide to adequacy of resuscitation is the hourly urine output, which should be maintained between 25 ml and 50 ml per hour. The patient with extensive burns (greater than 30 percent) should have an indwelling catheter and hourly urine output measured and recorded. Oliguria in the first 48 hours postburn, with the possible exception of patients with electric injury, is rarely caused by acute renal failure and is treated by increased fluid administration rather than by decreased fluid administration or diuretics.

Three categories of patients may require an osmotic diuretic: (1) those patients with significant electric injury in whom the occurrence rate of acute renal failure is approximately 20 percent, (2) those patients with associated crush or other injuries with extensive tissue death and large hemochromogen loads in the urine, and (3) those patients with large burns to whom one has given considerably more than the estimated fluid requirement but in whom oliguria persists. Osmotic diuretics, such as mannitol (see p. 111), will insure an adequate urinary output, but one must remember that even in hypovolemic

patients this occurs at the expense of blood volume. Urinary output in these patients is no longer a guide to the adequacy of resuscitation. Other diuretics, such as furosemide and ethacrynic acid, also have been used in burn patients.

Tracheostomy

The indications for tracheostomy are essentially those which exist in any other surgical patient; namely, (1) acute laryngeal or upper airway obstruction, (2) inability to handle secretions, and (3) associated chest wall injury. Severe smoke inhalation with respiratory insufficiency is another indication for tracheostomy. If the burned patient is to be evacuated and the attending medical personnel feel that the adequacy of the airway is at all questionable, tracheostomy should be performed before movement rather than risk the possibility of acute airway obstruction intransit. Three categories of patients are most apt to require tracheostomy on the basis of the indications listed: (1) patients with severe head and neck burns, (2) patients with steam burns of the face, and (3) patients burned in a closed space who have inhaled smoke and other products of incomplete combustion.

The severe chemical tracheobronchitis which results from inhalation injury may cause acute respiratory insufficiency. Such patients may have marked hypoxemia persisting for several weeks. Marked bronchospasm and frequent bouts of coughing are common and the patient may raise sputum containing carbonaceous material, confirming the diagnosis of inhalation injury. Conservative therapy with administration of humidified air or oxygen and nasotracheal aspiration, as indicated, are employed initially. The ability of the patient to clear his tracheobronchial tree and the quantity of endobronchial debris will determine whether bronchoscopy is necessary and the frequency with which it should be employed. A tracheostomy should be performed only if hypoxemia, bronchospasm, and endobronchial tissue desquamation cannot be controlled by conservative measures. After obtaining culture specimens from the tracheobronchial tree, a broad-spectrum antibiotic should be given; it then may be altered according to

the bacteriologic reports. Mucolytic agents and bronchodilators also may prove useful. Ventilatory assistance by mechanical means may be required in the treatment of those patients who have profound hypoxemia or severe bronchospasm. Steroids in large doses are employed only in those patients with unrelenting bronchospasm and progressive respiratory insufficiency.

Escharotomy

Circumferential full-thickness burns of the limbs may result in impaired circulation to distal unburned parts. To prevent secondary ischemic necrosis of these tissues, an escharotomy may be necessary to relieve constriction caused by edema beneath the inelastic, unyielding eschar. The procedure can be carried out without anesthesia in the ward or emergency room since it is performed through insensitive, full-thickness burn. The escharotomy incision should be carried throughout the entire length of full-thickness burns to insure adequate release of vascular compression. Swelling and coldness of distal unburned parts are not indications for escharotomy, but cyanosis, impaired capillary filling, and progressive signs of neurological damage are. Should evidence of vascular impairment be present, escharotomy is carried out in the midlateral line of the involved extremity and, if distal blood supply is not improved, a second escharotomy incision is made in the midmedial line in the longitudinal axis of the limb. Carrying the escharotomy across involved joints is critically important since in these areas of lesser subcutaneous padding the nerves and vessels are most easily compressed. The incision is carried through the eschar and the immediate subjacent thin connective tissue to permit expansion of the edematous subcutaneous tissue; if performed in this manner, blood loss is not excessive. Fasciotomy is rarely necessary and is required only in those patients with electric injury, other patients with particularly deep burns, or patients with associated traumatic injuries where edema exists below the investing fascia.

Patients with circumferential truncal burns also may require

escharotomies in the anterior axillary line to relieve restriction of chest wall movement by the eschar and permit a more satisfactory ventilatory exchange. This is particularly important in children with truncal burns who may be rapidly exhausted by the increased respiratory effort required. These patients frequently will be restless, agitated, and hypoxemic before escharotomy, and show prompt clinical improvement as well as improved ventilatory exchange and blood oxygenation following escharotomy. An incision along the lower margin of the rib cage may be necessary in those patients with deep burns extending onto the upper abdominal wall.

Adjuvant Treatment

The burn patient who has been actively immunized against tetanus should be given a booster dose of tetanus toxoid. Those patients who have not received prior active immunization should receive hyperimmune human antitetanus serum as well as an initial dose of tetanus toxoid, with active immunization continued at weekly intervals until complete.

Unless specifically contraindicated, penicillin is administered to all burn patients for the first 5 days postburn to prevent beta-hemolytic streptococcal burn wound infection. Thereafter, antibiotics are administered only on the specific indication of clinical sepsis supported by positive bacteriologic cultures.

As previously noted, restlessness and agitation frequently can be relieved by insuring adequate oxygenation, and the need for analgesia is usually minimal except in those patients with extensive partial-thickness burns. Analgesia, when required in the first 3 days postburn, should be administered intravenously in appropriate dosage.

Ileus is a common accompaniment of thermal injury of 20 percent or more of the total body surface, and nasogastric intubation and drainage to prevent emesis and aspiration are critically important in these patients. It is also important to maintain nasogastric intubation in all patients who are to undergo air evacuation, not only in the early postburn period but also later if evidence of gastrointestinal dysfunction exists.

WOUND CARE

Attention is directed to the burn wound after hemodynamic stability and the previously mentioned aspects of patient care have been accomplished. General anesthesia is not required for burn wound debridement; in fact, during this period of vascular instability and hypovolemia, it is ill-advised. Intravenous analgesia will suffice for pain control during such a procedure. The body hair is shaved from the areas of thermal injury and well back from the margins. The burns are gently cleansed with a surgical soap solution, and nonviable epidermal remnants are debrided. Bullae are excised since the proteinaceous fluid contained within them is an ideal culture medium for bacteria. After this initial debridement, the patient may be placed in bed, on surgically clean sheets. During the period of active wound exudation, placing bulky dressings beneath the burned parts to absorb the serous exudate has been found helpful. These dressings should be changed as necessary, and patients with circumferential burns should be turned to expose opposing surfaces every 3 hours.

Patients with burns of the buttock, perineum, and thigh do not require colostomies; the frequency of anal stricture is greatly increased by performance of such a procedure. Even should an abdominal operation be required to treat associated injuries, performing a colostomy is unwise solely because of the presence of buttock, perineal, or upper thigh burns. If a colostomy is indicated for other reasons, daily anal dilatations are mandatory.

Fractures associated with thermal injury are best treated by skeletal traction to permit exposure of the burns and their treatment with topical chemotherapy. The application of a cast over an area of thermal injury promotes suppuration and enhances the possibility of the development of burn wound sepsis. Nevertheless, plaster is acceptable over areas of burn in preparation for and during evacuation, if the cast is bivalved and removed promptly when the patient arrives at the definitive treatment installation.

ELECTRIC INJURY

Although the pathologic change resulting from electric injury is coagulation necrosis, the extent and severity of such injury may initially be seriously underestimated. Limited areas of cutaneous necrosis may be evident at points of entry, exit, or arcing, yet be associated with extensive, subcutaneous, deep tissue involvement, leading to inappropriate estimation of resuscitation fluid requirements. This "iceberg" effect also may necessitate the performance of fasciotomy rather than escharotomy to insure viability of distal unburned parts. The prophylactic use of an osmotic diuretic may be indicated because of extensive muscle necrosis with consequent liberation of hemochromogens. The presence of brawny, deep induration in a limb involved in electric injury with signs of vascular impairment indicates a need for fasciotomy. Approximately one-third of all patients with significant electric injury of the extremities will require an amputation of nonviable parts. This procedure should be delayed until resuscitation has been completed unless signs of systemic toxicity develop. Amputations in this situation as in any thermal injury should, if consistent with conservative principles of limb salvage, be carried out by disarticulation without opening a marrow cavity in the presence of the contaminated burn wound. Repeated debridement or reamputation is frequently necessary because of the progressive extension of tissue destruction, presumably from further small-vessel thrombosis.

CHEMICAL BURNS AND WHITE PHOSPHORUS INJURY

The depth and severity of chemical burns are related to both the concentration of the agent and the duration of contact with the tissues. These are the only burn injuries which require immediate care of the burn wound. The offending agent must be washed from the body surface as soon as possible. Full-thickness, third-degree injury of the skin caused by strong acids

may result in tanning or bronzing of the skin which will be waxy, yet pliable to the touch, leading the unwary to underestimate the extent of burn.

Many antipersonnel weapons employed in modern warfare contain white phosphorus. Fragments of this metal, which ignite upon contact with the air, may be driven into the soft tissues. Most of the cutaneous injury resulting from phosphorus burns is due to the ignition of clothing, however, and is treated as a conventional thermal injury. First aid treatment of patients with imbedded phosphorus particles consists of copious water lavage and removal of the identifiable particles, following which the involved areas are covered with a saline-soaked dressing, kept moistened until the patient reaches a definitive treatment installation. The wounds containing imbedded phosphorus particles then may be rinsed with a dilute (1 percent) *freshly* mixed solution of copper sulfate. This solution combines with the phosphorus on the surface of the particles to form a blue-black cupric phosphide covering which both impedes further oxidation and facilitates identification of retained particles. Under no circumstances should copper sulfate solution be applied as a wet dressing, and wounds must be lavaged thoroughly with saline following the copper sulfate rinse to prevent absorption of excessive amounts of copper. Copper has been associated with intravascular hemolysis of significant magnitude. An adjunct to the management of phosphorus injuries is the identification of the retained phosphorescent particles in a darkened room during debridement.

Combustion of white phosphorus results in the formation of phosphorus pentoxide, a severe pulmonary irritant. The ignition of phosphorus in a closed space may result in the development of concentrations of phosphorus pentoxide sufficient to cause acute inflammatory changes in the tracheobronchial tree. The effects of this gas can be minimized by placing a moist cloth over the nose and mouth to inactivate the gas and prevent endobronchial irritation.

Recently, hypocalcemia and hyperphosphatemia have been described as results of white phosphorus injury and have been associated with electrocardiographic changes. Sudden deaths in the early postinjury days suggest cardiac arrhythmia as a causative factor.

TOPICAL CHEMOTHERAPY

If the burn patient can be moved to a definitive treatment installation within 48 to 72 hours, no specific topical therapy need be employed in the field. However, if either the tactical or the logistic situation is such that treatment must be continued at a relatively forward area, topical chemotherapy should be begun once the patient has achieved hemodynamic stabilization. Sulfamylon (mafenide) burn cream should be applied in a layer one-eighth inch thick to the entirety of the burn wound with the sterile gloved hand immediately following initial debridement and wound care. Twelve hours later, to insure continuous topical chemotherapy, the $\frac{1}{8}$-inch-thick coat of Sulfamylon should be renewed to those areas of the burn wound from which it has been abraded by the bedclothes. Once each day, the Sulfamylon should be cleansed gently from all of the burn wound and the wound inspected by the attending physician. Daily debridement should be carried out to a point of bleeding or pain without employing anesthesia. Sulfamylon is an N^1-unsubstituted sulfonamide and produces inhibition of carbonic anhydrase leading to a mild bicarbonate diuresis with acidosis. This is usually of no consequence unless the patient develops pulmonary complications which cause retention of carbon dioxide. In the presence of a diminished serum bicarbonate, such a patient may quickly reverse from a state of alkalosis to one of acidosis even with a measured pCO_2 at what usually is considered a normal level.

Approximately 7 percent of patients treated with Sulfamylon develop some evidence of hypersensitivity in the form of a maculopapular rash usually appearing in the second week of treatment. In all but a few patients, this can be controlled readily by administration of an antihistamine, and Sulfamylon therapy may be continued. The acid pH and the hygroscopicity of the burn cream may cause considerable discomfort in areas of second-degree burn, and the patient with extensive second-degree burn involvement may require analgesia at the time of Sulfamylon application during the first 10 days of hospital care. Pain is not an indication for discontinuing Sulfamylon topical therapy.

If Sulfamylon is not available, a 0.5-percent solution of silver nitrate applied to the burn as wet soaks in the form of a multiple-layered gauze dressing changed twice each day and moistened every 2 hours is an accepted form of topical therapy. Losses of sodium, potassium, chloride, and calcium should be anticipated and appropriately replaced. Silver nitrate soak therapy is effective for bacterial control in burn patients who are received immediately after injury; but because of its lack of penetration of the eschar and underlying necrotic subcutaneous tissue, it is much less effective in the treatment of established eschar infection; an absorbable, topical medication is required in this case.

EVACUATION

The burn patient best tolerates movement by either ground or air in the early postburn period; that is, after hemodynamic and respiratory stabilization and before the development of septic complications which may make movement particularly hazardous. Patency of the airway must be insured throughout the evacuation procedure, and continued appropriate fluid administration via a secure intravenous pathway is essential. Nasogastric intubation with adequate gastric decompression is also necessary during patient movement in the early postburn period if any gastrointestinal dysfunction exists. Bulky dressings may be used effectively during evacuation.

It is essential that adequate documentation of the patient's premovement and in-flight course be maintained and accompany the patient so that continuity of medical care can be insured. Particularly important in this regard is an adequate record of administered fluids, urinary output, medications administered, and any other features of the patient's course that will require serial evaluation, such as neurological deficit. During evacuation, the seriously ill, extensively burned patient should be accompanied by trained surgical personnel familiar with the exigencies of patient movement during the early postburn course.

CHAPTER IV

Cold Injury

Although cold injury occurs sporadically in the civilian population and in peacetime, it is of primary importance to military forces. Larrey's description of the role which cold injury played in the defeat of Napoleon's army in Poland in 1812 is still a classic, as are his other descriptions of this condition.

Cold injury was a matter of most serious consequence during World War II in the Aleutians and in the Mediterranean and European theaters of operations. If the statistics are limited to hospital admission for the primary diagnosis of cold injury, the total figure for trenchfoot among U.S. forces is 57,504 cases (48,366 in Europe and 9,138 in the Mediterranean theater), and the total figure for frostbite is 12,639 (11,974 in Europe and 665 in the Mediterranean). During the war, 90,535 casualties with a diagnosis of cold injury were admitted to medical treatment facilities. This number included patients admitted for cold injury and those admitted for other causes but also suffering from cold injury.

In the Korean war, cold injury once again created a high morbidity. A detailed prospective epidemiologic study conducted during the Korean conflict furnished much of the information contained in the ensuing paragraphs.

CLASSIFICATION

Trauma produced by exposure to cold includes, in order of ascending seriousness, chilblain; trenchfoot and immersion foot;

frostbite, including high altitude frostbite; and total freezing. All of these conditions represent various degrees of a fundamental pathologic process which, irrespective of environmental and other modifying factors, are all related to the common factor of cold.

Although the distinction between the various types of cold injury is often artificial, particularly the distinction between trenchfoot and immersion foot, the following definitions are in fairly general use:

1. Chilblain, which frequently affects the hands as well as the feet, may result from exposure to air temperatures from above freezing to as high as 60° F (16° C), and often is associated with high humidity.

2. Immersion foot implies an injury caused by exposure, usually in excess of 12 hours, to water at temperature usually below 50° F (10° C).

3. Trenchfoot, which also may occur in the hands, results from prolonged exposure to cold at temperatures ranging from just above freezing to 50° F (10° C), often in damp environment, and usually in connection with immobilization and dependency of the extremities.

4. Frostbite implies the crystallization of tissue fluids in the skin or subcutaneous tissues after exposure to temperature of 32° F (0° C) or lower. Depending upon the ambient temperatures and wind velocity, the exposure necessary to produce frostbite varies from a few minutes to several hours. Frostbite may occur at various altitudes. Special attention has been given to high-altitude frostbite.

The ambient temperature decreases approximately 2° C for every 1,000 feet of increase in altitude. The temperature becomes stable at about −67° F (−55° C) at an altitude of 35,000 feet or above and exposure to these very low temperatures may result instantaneously in severe injuries to exposed parts of the body.

PATHOGENESIS AND PATHOLOGIC PROCESS

Pathogenesis

Trenchfoot, frostbite, and immersion foot are the cold injuries of greatest military importance. High-altitude frostbite may seriously disable aircrews in damaged aircraft. The nonfreezing effects of cold and their impact on digital dexterity and general function also may be a matter of consequence when aircrew compartments lose heat when operating at high altitude or at low altitude in cold environments.

The type of injury produced by cold is dependent upon the temperature at which the exposure occurs, the duration of the exposure, and the environmental factors which intensify the effect of the temperature. On most of these points, it is difficult to make positive statements. The major physiological protective mechanism against cold injury is believed to be the spontaneous cyclic rewarming of cold extremities which occurs as large-capacity arteriovenous shunts open in response to cooling (at a skin temperature of between 40° and 60° F) and allow relatively large volumes of blood flow through such cold injury-prone extremities as fingers, toes, and ears. This mechanism, known as CIVD (cold induced vasodilatation), may be enhanced by previous cold exposures, is much reduced or absent when the individual is thoroughly chilled, hungry, frightened, or tired, and tends to be less apparent in Negro and perhaps in other races.

The average duration of exposure in trenchfoot is usually 3 days, but the range is from a few hours to many days, with individual susceptibility apparently playing a considerable role. The average duration of exposure in frostbite is 10 hours but varies with ambient temperature, moisture, clothing, activity, and other factors to be discussed.

Pathologic Process

Although a number of physiologic changes induced by cold may explain tissue loss, it is doubtful that they all play a significant part in clinical cold injury. Intracellular molecular

changes due to hyperosmolarity, direct metabolic impairment secondary to the cold, and cellular structural damage from the mechanical effect of ice crystals seem far less important than impairment of nutritional blood flow as a final determinant of tissue injury after thawing. Vascular stasis following thaw from freezing injury has been well demonstrated. Clinical and experimental data indicate the importance of capillary blood flow as the determinant of reversibility in tissue freezing.

Alterations in capillary permeability are evident from experimental data and, clinically, from the edema and bleb formation that occur soon after thawing. Endothelial disruption may be responsible for the progressive capillary stasis, plugging, and thrombosis that eventually occur.

EPIDEMIOLOGY

Agent Factors

The military community responds to cold trauma according to accepted epidemiologic principles. The specific causative agent is cold. Moisture is closely related because it speeds the loss of body heat although it alone cannot cause cold injury. Cold produces injury by increasing the rate of body heat loss. This rate is determined not only by the ambient temperature, but also by other factors such as moisture and wind. Moisture increases the rate of heat loss by conduction and evaporation and wind by convection.

Environmental Factors

A variety of environmental and host factors combine in the total causation of cold injury and influence the incidence, prevalence, type, and severity of the injury, though this influence varies from situation to situation.

The most important environmental factors in cold injury are weather, clothing, and type of combat action.

Weather is a predominent influence in the causation of cold

injury. Temperature, humidity, precipitation, and wind modify the rate of loss of body heat. Low temperatures and low relative humidity favor the development of frostbite. Higher temperatures, together with moisture, favor the development of trenchfoot. Wind velocity and low temperatures, interacting, as expressed by wind chill, accelerate the loss of body heat under conditions of both wet and cold.

The type of combat action apparently is the most important environmental factor. Units in reserve or in rest areas have few cases. Units on holding missions or on static defense, in which exposure is greater, show a moderate increase in incidences. Factors which modify the incidence in relation to the rate of combat action include immobility under fire; prolonged exposure; lack of opportunity to warm the body, to change clothing, or to carry out measures of personal hygiene; fatigue; fear; and the state of nutrition. In warfare, in which exposure under conditions of stress may be prolonged, adequate clothing becomes essential to welfare and survival.

Host Factors

The following host factors influence the development of cold injury:

1. Age. There is no convincing evidence that age is a significant epidemiologic factor in cold injury among combat troops.

2. Branch of service. Trenchfoot and frostbite have a high selectivity for frontline riflemen, especially for riflemen of lower ranks. In World War II, approximately 90 percent of all casualties from cold occurred in riflemen.

3. Previous cold injury. Individuals with previous cold injury have a higher than normal risk of subsequent cold injury. The fact that such repetitive injury infrequently occurs at the same site suggests that this relates to individual susceptibility rather than a modification of circulation or local resistance to cold by the previous injury.

4. Fatigue. Both physical and mental weariness contribute to apathy which leads to neglect of all acts except those vital to survival. Fatigue is most evident in troops who are not rotated

and must remain exposed and in combat for long periods of time.

5. Other injuries. Concomitant injuries associated with reduction of circulating volume or localized reduction in blood flow predispose to cold injury. In addition, immobilization associated with concurrent injury increases the risk of frostbite in cold environments if adequate additional insulating protection is not available.

6. Discipline, training, and experience. Cold injury is preventable. Well-trained and disciplined men can be protected from cold injury even in adverse, pinned-down positions if they are knowledgeable concerning the hazards of cold exposure and informed regarding the importance of personal hygiene, especially care of the feet, exercise, and care of protective clothing. Such discipline and training are a *command*, not a medical, responsibility and reinforcement of these principles throughout a field operation is essential to the goal of protection from cold injury.

7. Psychological factors. Cold injury tends to occur in passive, negative, and hypochondriacal individuals. Such persons show less muscular activity in situations in which activity is unrestricted and are careless about precautionary measures when cold injury is a threat. Fear, by reducing the spontaneous rewarming known as CIVD, also may increase the incidence of cold injury.

8. Racial susceptibility. In all studies concerning World War II, Korea, and the recent experience in Alaska, the Negro had from four to six times the incidence of cold injury as did his Caucasian counterpart, matched for geographical origin, training, education, and so forth. This is not to say, however, that the Negro cannot be protected against injury. It does mean that he must be especially vigilant during cold exposure.

9. Drugs and medication. Any drug modifying autonomic nervous system responses, altering sensation, or modifying judgment can have disastrous effects on individual performance and survival in the cold. These factors must be impressed upon physicians involved in the care of patients in cold environments and must be impressed upon individual unit commanders and their men.

CLINICAL MANIFESTATIONS

Patients generally incur cold injury without being aware of it. They may have a first symptom of uncomfortable coldness but, subsequently, with continued heat loss, there is anesthesia of the affected part. Superficial cold injury, most commonly about the ears, may be associated with a tingling or stinging sensation. This sensation, however, frequently is not noted in freezing injury of the limbs. The part is without feeling, the patient complaining that it feels "like a stump" or "like a block of wood."

The first physical manifestation of cold injury is reddening of the skin which later becomes pale or waxy white. It is difficult to identify the degree of cold injury when the patient is first seen before, or soon after, rewarming. Any judgment is necessarily retrospective except at the two extremes of injury—very mild and very severe.

It is more realistic to separate cases into two categories—superficial and deep—although it is still instructive to review the classical description of the four degrees of injury. It should be kept in mind, however, that even in retrospect, there is overlap, and so-called second-degree injury of the digits may be associated with atrophy of the volar pad, hallmark of a deeper injury.

In first-degree cold injury, hyperemia and edema are early manifestations. The skin becomes mottled blue or purple, and then red, hot, and dry. Swelling begins within 3 hours and persists for 10 days or more if the patient is ambulatory; if the patient is kept at bed rest, the edema usually subsides in fewer than 5 days. Desquamation of the superficial layers of the skin begins within 5 to 10 days after injury and may continue for as long as a month. The toes may remain cyanotic, especially in the dependent position.

Some patients continue to complain of a deep-seated ache or paresthesia of the toes and ball of the foot. Severe paresthesia may appear within 3 to 13 days after rewarming. Hyperhidrosis and coldness of the injured part may appear 2 to 3 weeks after injury and may persist for many months. Frequently,

patients who have suffered first-degree cold injury will notice an increased sensitivity to cold in the affected part.

In second-degree cold injury, hyperemia and edema are also early manifestations. Edema is not marked and disappears within 3 to 5 days after rewarming if the patient is not ambulatory. Superficial vesicles appear within 12 to 24 hours of rewarming. If the hands are affected, superficial vesicles appear on the dorsum of one or more fingers. As the vesicles dry, within 12 to 24 days after rewarming, they form black eschars. There may be slight limitation of motion of the parts. The eschar gradually desquamates, revealing intact skin, which is, however, soft, thin, poorly keratinized, and easily traumatized. Throbbing or aching pain is noted 3 to 20 days after injury. Hyperhidrosis is apparent between the second and third weeks.

Third-degree cold injury involves the full skin thickness and extends into the subcutaneous tissues. The vesicles in third-degree injury are smaller and less tense, and may be hemorrhagic. This lesion contrasts rather strikingly with the blebs associated with less severe, second-degree injuries, where the blebs are frequently large and tense and extend to the tips of the fingers or toes, or to the base of the nails. Edema of the hand or foot may occur in third-degree injury although it usually disappears within 5 to 6 days. The skin overlying the area forms a black, hard, dry eschar as in second-degree injury. When the eschar finally desquamates, the remaining ulcer epithelializes if there is no complicating infection. Average healing time is 68 days.

Most patients complain of burning, aching, throbbing, or shooting pains, beginning between the 5th and 17th days and usually lasting from 2 to 5 weeks. Hyperhidrosis and cyanosis appear between the 4th and 10th weeks.

In fourth-degree cold injury, there is destruction of the entire thickness of the part, including bone, with resulting loss of all injured tissues. The part involved in fourth-degree injury is anesthetic; edema, however, may be present, depending primarily upon the manner in which the injury evolved. Vesicles, if present, are frequently small, scattered, and hemorrhagic. The tissue remains deeply cyanotic. In uncomplicated cases where freezing has been relatively rapid, loss of volume of the

affected part and progression to dry gangrene occur. With less rapid freezing, where some vascular inflow has remained in the early postthaw period, edema may be marked, and systemic toxicity without obvious local infection is common. The injured tissues become progressively black, dry, and mummified over a period of approximately 20 days. The line of demarcation becomes apparent on an average of 36 days and extends down to the bone between 60 and 80 days after injury.

MANAGEMENT

A major deterrent to evaluation of therapy has been the inability to predict the outcome in any given cold injury early in the postthaw period. Because of this, nuances of clinical management have been very difficult to evaluate. Since the extent of injury to the tissue is related to temperature and duration of exposure, rapid rewarming is of primary importance. Other therapeutic programs, including anticoagulant therapy, low molecular weight dextran or similar agents, or surgical or pharmacologic sympathectomy, while theoretically sound, and supported in some instances by experimental data, have not had controlled clinical trial sufficient to encourage their general use.

In the light of most clinical experience, it should be emphasized that meddlesome manipulations, rubbing, application of unguents, or exposure to excessive temperatures should be guarded against carefully. As soon as cold injury is recognized, every effort should be made to avoid compounding the effects of cold with physical injury.

In military operations, the treatment of cold injuries is influenced by (1) the tactical situation, (2) the facilities available for evacuation of casualties, and (3) the fact that most cold injuries are encountered in large numbers, during periods of intense combat, at the same time that many other wounded casualties also appear. Highly individualized treatment under these circumstances may be impossible. Examination and treatment of more life-endangering wounds must take precedence over this injury.

As a practical matter, any specific therapy designed to modify

the physiologic changes in cold injury must be instituted very early after thawing. Since, in many cases, the injury is not seen until some time after thawing, contemplation of therapy is purely academic and the major emphasis must be on protection from further injury, avoidance of premature surgery that might sacrifice otherwise viable tissue, early identification and control of infections, attention to maintenance of extremity function through early physiotherapy, and generalized nutritional support.

First Aid

The emergency treatment of cold injury is as follows:

1. All casualties with involvement of the lower extremities should be treated as litter cases if feasible.

2. Carefully assess concomitant injury or complicating systemic problems.

3. All constricting items of clothing, such as boots, gloves, and socks, should be removed if adequate protection from further cold exposure is available. Boots and clothing frozen on the body should be thawed by immersion in warm water before removal. Vigorous manipulation of frozen parts or attempts at range of motion or massage should be avoided.

4. If the injured parts are still frozen when first seen, they should be rewarmed rapidly by immersion in water at 100° to 104° F (37.5° to 40° C) with added antiseptic soap, such as pHisoHex, and with agitation of the water bath to hasten the warming. A whirlpool apparatus is most satisfactory for this.

5. General body warmth must be maintained. Sleep and rest should be encouraged.

6. A booster dose of tetanus toxoid should be given to those previously immunized. No indication exists that prophylactic use of antibiotics is valuable either in promoting healing or in preventing superficial or deep infection. In fact, the use of prophylactic antibiotics encourages emergence of resistant organisms.

7. Large vesicles or bullae should be protected and kept intact if possible. Once ruptured, it is usually desirable to debride the vesicle. Ointment dressings have no place in the usual

management of cold injury. Protective dry dressings are desirable during transportation, and sterile cotton should be used between the toes to prevent maceration.

8. Smoking is prohibited.

Later Management

When the patient reaches a hospital or a facility for definitive care, the following treatment should be employed:

1. Continued diligence to avoid injury of already compromised tissue should be maintained. In general, for lower extremity injuries, this is accomplished by keeping the patient at bed rest, with the part elevated on surgically clean sheets under a foot cradle and with sterile pledgets of cotton separating the toes. Weight-bearing on injured feet should not be allowed until mature epithelial tissue has developed over the affected areas. In upper extremity injuries, elevation is also desirable on sterile towels, with special care to avoid injury to bullae.

2. In an effort to reduce superficial bacterial contamination, the affected part is treated by whirlpool bath at 98.6° F (37° C), with pHisoHex added, on a twice-a-day basis, encouraging *active* motion on the part of the patient during the whirlpool treatment. Whirlpool baths assist in superficial debridement and make active range of motion exercises more tolerable to the patient and less traumatic to the tissues.

3. Analgesics may be required in the early postthaw days, but continued requirement for analgesics in uncomplicated injuries is uncommon.

4. The patient should be encouraged to take a nutritious diet with adequate fluid intake to maintain hydration.

5. Patients should be placed on surgically clean sheets and all lesions exposed to the air at normal room temperature.

6. Superficial debridement of ruptured blebs should be performed, and suppurative eschars and partially detached nails should be removed. Close attention should be maintained to circumferential eschars or eschars where vascular compromise could be a problem. Such eschars at least should be bivalved, although complete debridement is occasionally necessary. In the management of cold injury, early amputation has no place.

Surgical intervention should be deferred until a distinct line of demarcation has developed. There is usually healthy granulation tissue under an eschar at the line of demarcation. Delay of surgical procedures, especially in upper extremity injuries, will enhance the potential for a functional result. Rarely, generalized sepsis from large areas of necrotic and infected tissue will necessitate amputation. Skin grafting, while not a function of forward facilities, is occasionally indicated to protect denuded areas over vital structures.

Active physiotherapy should be instituted during daily whirlpool as soon as possible.

Newly epithelialized areas are susceptible to minor trauma, as in walking, and are especially sensitive to cold. Therefore, continued protection must be offered until normal keratinization has occurred. Subsequently, special skin care may be required to deal with residual hyperhidrotic states.

PROPHYLAXIS

The successful prevention and control of cold injury depend, first of all, upon vigorous command leadership, the provision of adequate clothing, and a number of individual and group measures. These measures include:

1. A thorough appreciation and comprehension by command, staff, technical personnel, and all combat components of the potential losses which may occur from cold injury, both in winter combat and in other circumstances in which cold injury has been known to occur.

2. Full command support, by echelon, of a comprehensive and practical cold injury prevention and control program. It should be emphasized again that this is a *command,* not a medical, responsibility.

3. Indoctrination of all personnel in the prevention of cold injury individually and by units.

4. The provision of adequate supplies of clothing and footgear and their correct utilization to avoid exposure to cold. The program of supply must provide adequate dry clothing for the daily needs of the soldier who is farthest forward in combat; it must also provide for the correct fitting of clothing

and boots. All articles of clothing must be sized and fitted to avoid constriction of the extremities and tightness over the back, buttocks, and thighs.

Clothing for cold weather, based on the layering principle, is now designed as an assembly for protection of the head, torso, and extremities. The clothing is worn in loose layers, with air spaces between the layers, under an outer wind-resistant and water-resistant garment. Body heat is thus conserved. The garment is flexible, and inner layers can be removed for comfort and efficiency in higher ambient temperatures or during strenuous physical exertion. Prevention of loss of body heat by proper protection of the body is equally as important as the efficient use of appropriate dry footgear and warm dry gloves. Finally, the most efficient clothing is of no value unless, through training, a high level of individual and unit foot and clothing discipline are maintained.

5. Special protection for racial and other groups who are especially susceptible to cold injury, together with the regular rotation of all troops. It should be remembered that patients with exposed wounds and injuries are particularly liable to cold injury because blood and transudate from their wounds will freeze from the clothing inward.

6. Effective policies of sorting in forward areas, with provision for early evacuation and treatment of casualties actually suffering from cold trauma.

7. The identification of factors responsible for cold injury in special situations, which is a command responsibility. Significant numbers of cases occur as a result of barehanded contact with cold metal or gasoline; as a result of rapid deployment of troops seated in unheated vehicles, without interruption for short, rewarming marches every few hours; as a result of airdrops of troops into cold areas without adequate protective equipment and training; or as a result of several hours' confinement of arctic-equipped airborne troops in heated aircraft, followed by a drop into a subzero environment after their insulating clothing has been saturated with perspiration. Only by the evaluation of these factors can the specific measures necessary in particular units or groups be put into effect.

CHAPTER V

Blast Injury

Blast injuries result from the energy released when pressure waves generated by an explosion strike body surfaces. The pressure or blast wave is transmitted through gas, liquid, or solid. Since the blast injury potential is directly proportional to the magnitude of the exploding force, the increased effectiveness of modern munitions has increased the lethality and the magnitude of such injuries in numbers and severity. Structures most frequently injured by blast are tympanic membranes, chest wall, lungs, abdominal wall, and viscera. Patients exposed to a blast force may be in a state of shock, without evidence of external injury. It is possible, therefore, that the victim of blast injury may be overlooked and may be required to perform feats of endurance for which he is wholly unfit and which may cause him to sustain serious and irreparable damage.

TYPES OF BLAST INJURY

Air Blast

The effects of air blast vary with the wavelength. In a blast which produces short wavelengths, evidenced by a clapping high-pitched sound, several waves will pass through the body in a given time and the possibility of rupture of a viscus or of other internal injury will be greatly increased. If the wavelengths are longer, manifest by a thundering low-pitched sound, only a single wave may pass through the body at a given time,

and the possibility of sudden disruption of internal organs is considerably less.

The tympanic membrane ruptures at a pressure of 7 pounds per square inch above atmospheric pressure. With this exception, the body can withstand a pressure of up to 30 pounds per square inch. This fact is especially valid if the blast waves are of low frequencies. If, however, the victim is in contact with a solid object at the time of the air blast, the blast wave transmitted through the body from the solid object can produce severe damage at far less pressure than that which is necessary to produce a similar injury in the open. Although a man might withstand a pressure of 30 pounds per square inch of body surface, he could be injured severely if he were knocked off his feet and thrown against a solid structure by the blast wave.

Underwater (Immersion) Blast

Explosive forces under water can cause grave injuries since pressure waves in water travel more rapidly than pressure waves in air and are effective at much greater distances. The human body has approximately the same density as has water, and the blast wave is transmitted through solid tissues without displacing them. When, however, the blast wave impinges on gas-filled cavities, such as the lungs and the intestines, its local effects may be enormously disruptive, even though parts of the body may be out of the water. The resulting injuries are chiefly in the abdomen and the chest.

Solid Blast

Solid blast injury is produced by the transmission of pressure waves through solid objects, such as the deck of a ship or the wall of a tank, sometimes without disruption of the solid object itself. These pressure waves may produce multiple fractures, disruption of major blood vessels, or damage to internal organs remote from the point of contact. The injuries may occur without damage to the skin.

CLINICAL MANIFESTATIONS AND DIAGNOSIS

If other injuries caused by primary and secondary missiles are excluded, the severity of injuries caused by blast is directly related to the distance of the victim from the explosion. Individuals near the center of the explosion usually will be killed instantly while those at a greater distance may survive.

The victims may not present external evidence of injury, but they are usually nervous, apprehensive, and tremulous, and an incorrect diagnosis of battle neurosis may be made. In such circumstances, patients improperly may be returned to their units or treated as walking wounded until shock, dyspnea, or other symptoms and signs indicate the gravity of their condition. Patients who have sustained other obvious injuries, such as open fractures, may not be recognized as having serious lesions caused by blast. If a general anesthetic is administered or a rapid transfusion is given in this clinical setting, the consequences may be dire if "blast lung" coexists with more obvious lesions. From such admonitions, it is clear that a detailed history of the wounding event, when possible, and evaluation, particularly of lung and intestine, are important.

It is also important to realize that such conditions as crush injury (see pp. 203–206) and carbon monoxide poisoning may be present in the same situations in which blast damage is sustained.

REGIONAL INJURIES

Ear Injuries

The low blast-wave pressure which ruptures the tympanic membrane, and the high incidence of explosions which generate a relatively low blast pressure, such as air bombardment and small arms and artillery fire, result in the ear's being the organ most frequently injured by a blast force. Blast injury to the ear may result in hemotympanum, rupture of the tympanic membrane, or discontinuity of the ossicular chain. The injury

may be unilateral or bilateral and is frequently associated with a hearing loss.

When rupture of the tympanic membrane is painless and there is no external bleeding, otoscopic examination may be necessary to demonstrate the damage. Other forms of blast injury may be present.

Treatment consists of gentle cleansing of the auditory meatus with dry, sterile cotton wool. The ear is not packed, and syringing and the use of ear drops are both contraindicated. Patients with rupture of the tympanic membrane should be evacuated since this injury may be associated with more severe damage to the middle ear.

Chest Injuries

Blast injuries of the chest include damage to the chest wall with rupture of, and hemorrhage into, pulmonary alveoli. These extremely dangerous injuries are the result of the direct impact of the ribs on the lung after the chest wall has been driven inward as the result of a nearby explosion. Bronchi, pulmonary alveoli, and capillaries may be ruptured by a blast wave transmitted through tissues without damage to the chest wall. Death may be instantaneous, but casualties with less severe injuries may survive.

Clinical manifestations of blast injuries of the lungs include shock, restlessness, cyanosis, rapid pulse, pain in the chest and upper abdomen, dyspnea, frothy hemoptysis, diminished respiratory excursions, and ineffective cough. Later, moist rales are demonstrable. Patients with pulmonary involvement are particularly likely to be considered as suffering from acute battle neurosis because they are extremely apprehensive and tremulous.

Objective signs may be slight. Rib fractures may be present. Injury to the intercostal nerves may cause abdominal rigidity and lead to a suspicion of intraperitoneal injury. Slight hemoptysis caused by multiple pulmonary hematomas is sometimes noted, but the finding of frothy, bloodstained mucus is more characteristic, particularly when noted in a patient with an ineffective cough mechanism. Not all of the symptoms and signs listed are likely to be present in a single patient.

Within a few hours of the injury, roentgenograms will show diffuse mottling in the affected lung fields. The differential diagnosis includes aspiration, pulmonary edema, and contusions and hematomas attributable to mechanisms other than blast.

The patient with a blast injury of the lungs must be treated immediately as a litter patient. Oxygen is administered for signs of respiratory distress. Positive-pressure oxygen, preferably by a volume respirator, may be indicated. Since pulmonary edema must be anticipated, the administration of blood, colloids, and other parenteral fluids is hazardous. If special indications exist for their use, the risk must be taken, but the rate of administration ordinarily should be slow and should be monitored by central venous pressure determinations and urinary output. Atropine may be used in small doses to diminish secretions. If these secretions are excessive, aspiration of the bronchial tree is performed as indicated and endotracheal intubation or tracheostomy may be necessary. Pulmonary edema may require the use of diuretics, and appropriate antibiotics are administered to prevent superimposed pneumonitis. Other injuries are cleansed and dressed, but debridement of wounds and other operative procedures must be planned carefully.

The choice of anesthesia techniques requires mature and experienced judgment. Although regional anesthesia, including nerve blocks, epidural or subarachnoid blocks, and local infiltration are desirable, inhalation endotracheal anesthesia may become necessary. Special precautions, however, involving light anesthetic planes and mechanical positive-pressure administration of agents, such as nitrous oxide or Fluothane, are required. Muscle relaxants and analgesic agents are used as supplements.

Patients with blast injuries of the chest should not be evacuated in less than 48 hours unless the tactical situation permits no other course.

Abdominal Injuries

Blast injuries to the abdomen produce injuries equivalent to blunt trauma of an equal force. Depending upon the force

exerted by the blast wave upon the abdominal parietes and viscera, the injuries incurred may vary from superficial contusions to multiple intra-abdominal organ injuries, which, in turn, may vary in severity from a minor subserosal hemorrhage to complete organ disruption.

The patient's history and clinical manifestations vary with the organ injured and the degree of injury. A common description of the abdominal pain associated with a blast injury to the abdomen is "like a kick in the belly." There is often a remission of the initial pain followed by recurrence. Other symptoms include nausea, vomiting, and an urge to defecate. Signs consist of diffuse tenderness, guarding or rigidity of the abdominal muscles, and pneumoperitoneum as manifested by loss of liver dullness.

Patients who survive the initial injury show various clinical manifestations. In mild cases, there is abdominal pain, with some tenderness on palpation and perhaps slight distention. Other patients complain of moderate colicky abdominal pain, associated with vomiting and frequent bowel evacuations. In this group, symptoms and signs usually subside within 48 to 96 hours. The transient pareses of the limbs which have been described in association with blast injuries of the abdomen are probably caused by minor vascular disturbances in the spinal cord. Melena or hematuria may accompany more severe injuries. In abdominal contusions, pain may subside promptly, but intraperitoneal abscess or fecal fistula may become manifest later. If primary perforation occurs at the time of injury, abdominal rigidity may be severe and shock may ensue. In cases of delayed intestinal perforation or secondary peritonitis, there may be no evidence of an intra-abdominal catastrophe for several days. Tenderness and pain in the upper abdomen after a blast injury may be due to chest injury whether or not an abdominal lesion is present. When clinical signs remain localized to the upper abdomen, they are probably of thoracic origin. When clinical signs, initially confined to the upper abdomen, spread downward to the lower abdomen, an abdominal injury is almost certainly present. When clinical signs are most marked from the onset in the lower abdomen, there is little doubt of intraperitoneal damage. Bright-red rectal

bleeding is evidence of injury of the large bowel or the rectum.

Exploration of the abdomen is generally desirable or essential with the following indications:

1. Suspected perforation of a hollow viscus with evolving peritonitis manifested by severe, unremitting, or increasing abdominal pain and tenderness. Absent bowel sounds, elevated pulse, rising temperature, and leukocytosis support this diagnosis.
2. Tenderness over the lower abdomen, especially associated with the presence of bloody stools.
3. Pneumoperitoneum, as manifested by loss of liver dullness or the roentgenologic demonstration of free subdiaphragmatic air.
4. Evidence of intraperitoneal bleeding.

Generally, exploratory laparotomy is indicated upon suspicion of intra-abdominal injury. If the tactical situation or other unusual circumstances preclude laparotomy, nonoperative therapy to include nasogastric suction and intravenous fluids should be instituted. Lastly, morphine and other sedation may be used after the course of therapy has been determined. These measures are employed to minimize the adverse effects of visceral rupture and peritonitis.

PREVENTIVE MEASURES

Blast injuries may be reduced in severity by the use of certain protective measures. Earplugs may be used to prevent injury to the eardrum. Body armor or padded clothing will protect the trunk. Guncrews may be protected by the use of baffle plates. The effects of exposure to underwater blast are minimized by swimming on the back or floating supine.

CHAPTER VI

Chemical Injury

During wartime, no matter what the area, the possibility of attack with chemical agents always exists. The further possibility thus exists that casualties may be suffering from the effects of chemical agents as well as from wounds. Only the most important principles concerning the treatment of casualties from the various chemical agents are described in this handbook.[1]

Many chemicals that can be used in warfare produce immediate symptoms and are quickly recognized, but others, depending upon their concentration and the length of exposure to them, may not become manifest for a period of time. The vesicants, for instance, may be overlooked immediately after exposure, but unless their presence is recognized and decontamination is carried out before any surgery is attempted, wound healing is seriously interfered with. The lung damaging agents, which are also slow to be recognized, greatly increase the hazard of anesthesia. Finally, failure to recognize chemical agents and to decontaminate the patient promptly constitutes a serious hazard to personnel who must handle him.

Chemical agents are classified on the basis of their physiologic action as follows:

1. Nerve agents.
2. Vesicants (blister agents).
3. Irritants (tear and nose agents).

---

[1] For details, see the NATO Handbook on the Medical Aspects of NBC Defensive Operations, AMedP-6 (Army Field Manual 8-9; Navy Publication NAVMED P-5059; Air Force Pamphlet 161-3), 31 Aug. 1978, part III.

4. Cyanogen agents.
5. Lung damaging agents (choking agents).
6. Incapacitants.
7. Other agents.

GENERAL PRINCIPLES OF MANAGEMENT

Medical officers must learn to identify chemical agents immediately from their effects upon man. The treatment of chemical agent casualties must begin at once. When, however, chemical casualties are received at a medical unit, they also may have traumatic lesions or illnesses from other causes. These casualties must be managed so as to minimize the injuries resulting from chemical exposure without aggravating the traumatic lesions or the other illness.

The most frequent problem is the determination of treatment priority among the surgical lesion, the medical condition, or the chemical injury. Measures to control emergency conditions, such as hemorrhage, shock, or respiratory problems resulting from causes other than exposure to a chemical agent, may be of equal or greater urgency than treatment for chemical poisoning. All required treatment and care measures, therefore, may have to be performed in rapid sequence or by simultaneous team action.

When a chemical casualty has associated injuries, the recommended order of priority in which emergency action should be taken is as follows:

1. Control of respiratory failure (assisted ventilation) and massive hemorrhage.

2. Administration of antidote, such as atropine for nerve agent, and nitrite for cyanides.

3. Decontamination of the face, and of the protective mask if its continued use is necessary.

4. Removal of contaminated clothing as soon as feasible and always before the casualty is moved indoors. Decontamination of skin, where required.

5. Additional emergency medical care and treatment for shock, wounds, and illness which is so severe that delay may endanger life.

6. Supportive medical care and treatment for less urgent wounds and other injuries or illnesses.

7. Transportation of the patient to an uncontaminated area for continued treatment.

NERVE AGENTS

Nerve agents rapidly penetrate all mucous surfaces; the vapor is quickly absorbed by both upper and lower respiratory tracts and by the cornea. The agents may be absorbed through the skin in either liquid or vapor form. They are almost odorless, effective in low concentrations, and difficult to detect by the senses. Nerve agents in any form can act very rapidly, and when the dosage is relatively heavy, convulsions and death may occur. The development of toxic symptoms, however, may be delayed for some hours after percutaneous poisoning with liquid agents. After extremely low doses of vapor, constriction of the pupils of the eyes and running of the nose are the two most common early effects. Low doses cause, among other effects, a feeling of constriction of the chest. After large doses of vapor, preliminary symptoms are followed rapidly by irregular and shallow breathing, slowing of the heart, possible convulsions, and death. If the dose is very large, death from absorption of vapor may occur in a matter of minutes. With percutaneous poisoning, there are no early changes but eventually a vague malaise develops, followed by nausea and vomiting, impaired breathing, possible convulsions, and death. The protective mask gives full protection to the eyes and respiratory tract, but special clothing is needed to protect the skin.

The four cardinal principles of therapy for nerve agent intoxication are:

1. Assisted ventilation.

2. Cholinergic blockade (atropine: dose depends on symptoms; mild to moderate symptoms will require about 2 mg I.M. initially, up to 6 mg in 25 min).

3. Decontamination.

4. Enzyme reactivation with an oxime.

VESICANTS (BLISTER AGENTS)

Vesicants, in both the liquid and the vapor state, may cause immediate or delayed irritation and burning to a varying degree of those parts of the body with which they come in contact. The conjunctiva and cornea of the eye are most sensitive, followed by the respiratory tract and moist skin. Dry skin is somewhat more resistant. "Mustard gas" has a faint smell and does not cause any immediate irritation to the eyes, lungs, or skin, even in dangerously high vapor concentrations. The tissue destruction usually is not visible for some hours. These insidious qualities make it a very dangerous agent. Death may occur from inflammation of the respiratory tract, the aftermath of extensive burning, superimposed pneumonia, or cytotoxic effects when the agent has been absorbed. The protective mask gives full protection to the eyes and respiratory tract, but special clothing is required to protect the skin.

IRRITANTS (TEAR AND NOSE AGENTS)

Although many compounds as vapors, smokes, or fine dusts affect the eyes and upper respiratory tract, certain of them in particularly low concentrations have a pronounced and immediate action causing spasm of the eyelids and lacrimation, severe burning in the nose and chest, violent coughing which may result in vomiting, and often profuse salivation. The skin may also smart if it is wet. The concentration required to produce these effects in the field does no permanent harm to the eyes or the respiratory tract. The agents are readily detected by their effects. Relief may be accomplished by removing the individual to fresh air or donning the protective mask.

CYANOGEN AGENTS

Cyanogen agents cause little or no local injury, but produce their effects after absorption into the body. The most important are hydrogen cyanide (prussic acid) and cyanogen chloride.

Hydrogen cyanide in high concentrations causes rapid death by tissue anoxia. Although this highly toxic gas did not prove a success in World War I, it still constitutes a potential danger.

Cyanogen chloride has a similar action to that of hydrogen cyanide. It is not lethal at low concentrations, but has both lacrimatory and choking effects. The protective mask protects the eyes and respiratory tract against these agents.

The diagnosis is made by the history of exposure, the bitter almond odor of cyanogen, and the bright-pink color of the skin. The success or failure of treatment of acute cyanide poisoning depends upon the speed with which cellular oxygen utilization can be restored. This may be facilitated by the production of methemoglobin which combines to form nontoxic cyanmethemoglobin. The introduction of thiosulfate assists the detoxification to harmless thiocyanate. The definitive treatment is controversial. The following measures have been suggested:

1. Ampoules of amyl nitrite are crushed in the hand and applied to the patient's nose. If gas is still present, the ampoules are inserted inside the facepiece of the mask. The original dose of two ampoules is repeated three times (eight ampoules).

2. If exposure has been severe, a 3-percent solution of sodium nitrite is given in 10-ml doses over a period of 1 minute until a total of 50 ml has been given. Fifty ml of a 25-percent solution of sodium thiosulfate is given intravenously also over a period of 1 minute between injections of sodium nitrite.

3. The administration of 100-percent oxygen has been shown to be effective.

LUNG DAMAGING AGENTS (CHOKING AGENTS)

Lung damaging agents are nonpersistent gases which cause pulmonary edema and asphyxia. The principal representative of this group is phosgene, which can be detected by its characteristic odor of new-mown hay in concentrations which can be inhaled without danger. Troops trained in the use of their protective masks will be fully protected, but heavy casualties might occur if these agents were to be used against civilians

who have no masks or have had inadequate training in their use.

If there are symptoms of respiratory distress after known exposure to phosgene, the casualty must be evacuated by litter, and absolute rest must be enforced while he is observed carefully to detect the possible development of massive pulmonary edema, which may appear suddenly. In severe injury, he is kept comfortably warm. Anoxia is treated by the use of oxygen and assisted ventilation when necessary. Sedatives are used sparingly to avoid depressing the respiration. Antibiotics may be used to prevent pneumonitis.

INCAPACITANTS

The incapacitating agents—such as the glycolates—produce mental and physical incapacitation. In minute doses, they will merely give changes in mood, varying from an apparent drunken happiness to deepest despair; in higher doses, they produce muscular weakness, hallucinations, and delirium, so that a man no longer knows who he is, or what he is doing. Their military effect, therefore, varies from the disturbance of morale to the complete breakdown of military discipline with inability to carry out orders. Onset may be delayed from minutes to several hours, and the duration of effects also varies with the agent used. With the slower acting compounds, there may be a period of physical incapacitation before mental incapacitation becomes apparent.

Incapacitants are active in very small amounts, and as such, they could be used in a covert role in water or food and may not be detected; in the overt role, they can be disseminated as smokes or dusts; some are liquids and may be absorbed through the skin.

Effects may last from minutes to hours, but recovery is complete without medical treatment. However, antidotes are available that greatly reduce the recovery time in returning the casualty to normal. Physostigmine salicylate, 0.06 mg per kilogram of body weight given intramuscularly, has been an effective antidote for the glycolate group of these agents. Protective

masks give full protection to the eyes and respiratory tract, but special clothing is needed to protect the skin.

OTHER AGENTS

This group contains substances which are toxic and might affect a man in battle as well as in ordinary military training or life. Substances considered are smokes, incendiaries, other noxious hazards such as nitrous fumes, and carbon monoxide.

It must be borne in mind that all smokes are harmful and that the use of smokes in confined spaces is very dangerous. Their inhalation may result in serious respiratory irritation and may lead to pulmonary edema. Some types of smokes containing ammonium chloride for cooling purposes will produce hydrogen cyanide and are most dangerous. Treatment for smoke inhalation is the same as for lung damaging agents.

Carbon monoxide poisoning should be suspected in any unconscious casualty from a poorly ventilated area. Such casualties may present with a characteristic cherry-red skin coloration. They should be treated with oxygen and assisted respirator ventilation if necessary.

CHAPTER VII

Mass Casualties in Thermonuclear Warfare

GENERAL

Nuclear weapons range in size from very small, not many times larger in total energy yield than the largest conventional bombs, to immensely large, the so-called thermonuclear or hydrogen bombs with yields in the megaton range. Total energy yield of nuclear weapons is rated in terms of equivalent amounts of TNT. Therefore, a weapon with a 20-kiloton yield has the same total energy output as 20,000 tons of TNT. A 1.0-megaton weapon has the energy output of 1 million tons of TNT. Energy is released by nuclear detonations in three forms: thermal radiation, blast, and ionizing radiation. The relative importance of each in causing casualties depends primarily upon three factors: the yield of the weapon, the environmental conditions in which the detonation occurs, and the distribution of troops in the target area. The thermal output can be the most significant casualty producer, particularly for the larger weapons. Blast, however, will produce nearly as many casualties, and blast and thermal injuries together will account for most of the casualties under almost all circumstances. Radiation, either at the time of detonation or later from fallout, also will be a significant injury-producing agent.

The use of nuclear weapons alone or in conjunction with other weapons can result in essentially instantaneous production

of very large numbers of casualties. When this occurs, the whole medical evacuation and treatment system is severely overburdened and some system of classification and sorting of casualties must be added to the normal procedures of evacuation and hospitalization. In addition, a system must be established to hold casualties who are too seriously injured to remain with their units but who need not or cannot be hospitalized. These two procedures, sorting of casualties and holding of the excess numbers, must be planned for as part of the normal organization and operations of the medical support system in a theater of operations.[2]

In applying the principle of providing the greatest good for the greatest number in the management of mass casualties, a field medical system must face and solve several problems. The location and number of casualties must be determined. This requires intact communications, since, on a dispersed battlefield, isolated units could suffer severe damage and many casualties and be unable to notify higher headquarters. Subsequent delay in initiating hospitalization and treatment will result in greatly increased morbidity and mortality.

The casualties must be evacuated. In frontline areas, enemy action exploiting the use of nuclear weapons could greatly hinder or prevent evacuation. In rear areas, adequate evacuation means may not be available to handle the massive number of casualties produced by an attack. The availability of helicopters would help since they could be diverted for use from one area to another much more readily than ground transportation. The use of nonmedical transportation systems may be required but cannot be planned on.

During the actual evacuation of large numbers of patients, *patients must be classified and selected* to insure that those who will benefit most from hospitalization will receive it first. The principles of sorting, as described in chapter XII, are applicable to determining priority of treatment generally. However, sorting at the site of a detonation often will be done by inexperienced and untrained personnel. Therefore, relatively simple guidelines must be established to aid them in determining

---

[2] For a detailed discussion of medical operations in nuclear war, including protective measures, see part I of footnote 1, p. 55.

priority of evacuation. Basically, the three groups of patients are:

1. Those with minimal injuries which do not incapacitate them completely and are not a significant threat to life. These casualties could continue as at least partially effective soldiers and would not qualify for immediate or early evacuation.

2. Those with severe multiple injuries who obviously are going to require extensive, time-consuming care. These also would be delayed.

3. Finally, those with relatively simple injuries which require immediate surgical treatment but are not extensive or multiple. These would get first priority for evacuation.

Eventually, however, all casualties unable to continue as effective soldiers will have to be evacuated. Further classification of patients will not be required for primary sorting for evacuation.The presence or absence of radiation injury, in general, will be ignored in preliminary sorting, since there are no reliable guidelines to aid in the early diagnosis of radiation injury.

As noted, there should be appropriate treatment facilities to which patients who cannot be treated immediately or who require only minimal treatment can be evacuated. These facilities should be set up with limited equipment and staffed with small numbers of medical personnel and should be part of the expansion plans of all field hospitals regardless of size or location. They should be as close to hospitals as possible so that the patients held there can receive appropriate additional care quickly and easily as the overall situation and balance between medical resources and patient load change. A great variety of patients, including those not fit for field duty but not requiring full-care-type hospitalization as well as the very severely injured, should be kept there. These should include patients in the following categories:

1. Minimal burns.
2. Mild trauma cases.
3. Mild chemical injury cases.
4. Severely injured patients who are not expected to survive and for whom treatment is not immediately available, but for whom supportive measures may be enough to keep them alive until treatment does become available.

Contamination of patients and equipment with radioactive fallout may occur frequently. Decontamination procedures must be part of every medical facility's standard procedures and must include provisions for disposal of the contaminated waste in small quantities by some procedure, such as burying, to avoid accumulation of radioactive materials to hazardous levels. The decontamination of patients must proceed along with resuscitative treatment and must not delay needed treatment. Decontamination can be incorporated into standard admission procedures and can be limited to simple removal of clothing and washing contaminated particles from exposed surfaces. The hazard to attending personnel is negligible.[3]

MEDICAL EFFECTS OF NUCLEAR WEAPONS

Proper management of the casualties of nuclear war requires an understanding of the medical problems to be expected. Nuclear weapons are sufficiently different in their casualty-producing potential from conventional weapons that the types of injuries will be different. It is important to understand these differences if sorting and medical treatment are to be accomplished effectively and quickly.

THERMAL BURNS

The extremely high temperatures produced by a nuclear explosion cause release of a large part of the energy in the form of thermal radiation. This radiation travels at the speed of light and is capable of producing severe burns at great distances. In nuclear combat, burn casualties consistently will make up a large fraction of the patient load, and a field medical service must plan for the increased logistic burden inherent in the requirement to manage thousands of burn cases on a continuing basis.

A major problem will occur during the initial evacuation of burn patients when massive numbers of casualties must be

[3] For details of hospital operations in fallout contaminated areas, see part I of footnote 1, p. 55.

handled. Sorting will be essential to conserve medical resources and should be done in accordance with the following criteria:

1. Cases involving 20 percent or less of the body surface should be treated on an outpatient basis or at minimal care facilities. These patients can care for themselves with minimal supervision. They will not be fit for duty and should not stay with their units if actively engaged in combat.

2. Patients with more than 20 percent body surface involvement or with associated blast injuries will require hospitalization for resuscitative treatment and surgical care.

3. Patients whose burns involve certain critical areas, such as the head and neck, hands, or feet, will require hospitalization even if the total body surface involvement is less than 20 percent.

4. Cases with more than 50-percent involvement have a decreasing chance of survival with increasing degree of involvement, and should be given a low priority for needed surgical care. They should be retained in a delayed status in the minimal care section of a medical facility where they will be available for more extensive treatment if resources and time allow. It must be realized that young healthy adults, without other injury or disease, can survive such burns with adequate treatment so that a rigid classification system denying them available treatment is not desirable.

All patients should receive as much treatment as possible and the above criteria must not be inflexible. However, any treatment must be accomplished as efficiently and quickly as possible, and long, time-consuming procedures may have to be delayed, or not done. The greatest good for the greatest number is best achieved by treating each patient as quickly and simply as possible, doing, first, what is essential to save his life, then what may be possible to save limbs, and, last, what might be required to save and restore function.

A small but significant percentage of burn patients will have associated radiation injury and will develop bone marrow depression during the course of their illness. These patients cannot be recognized upon admission since the bone marrow depression does not become clinically evident until after a latent period of 2 to 6 weeks after radiation exposure. A blood count in such cases during the first few days after exposure will show

a variable leukopenia, particularly of lymphocytes. These patients will have high morbidity and mortality rates due primarily to infection. Unless the procedures are required to save life, these patients should not be subjected to surgery during the phase of bone marrow depression. They should be treated with antibiotics and whole blood, preferably fresh, as needed until bone marrow recovery is evident. Necessary surgical procedures then can be performed, since essentially normal healing of the burn wounds is possible after marrow recovery. If there is no evidence of bone marrow recovery, the patient will not survive the present techniques of treatment available in the field.

BLAST INJURIES

Blast injuries caused by nuclear detonations are of two types: direct, due to overpressure effects (see p. 48), or indirect, due to drag forces of the winds accompanying the blast wave. This latter category includes a wide variety of missile and translational injuries.

Direct blast injuries will be rare since persons close enough to the point of detonation to sustain significant direct overpressures will almost invariably sustain lethal thermal and indirect blast injuries. However, those few patients who might be seen with direct blast injuries should be managed the same as any other direct blast injury. Their injuries will be complicated by other injuries and they will suffer a high incidence of radiation-induced bone marrow depression during their post-injury phase, resulting in increased morbidity and mortality. Direct blast-induced internal injuries easily can be overlooked in a mass casualty situation.

The blast wave of a nuclear detonation is unlike conventional blast waves in that its formation is associated with the production of severe, transient winds from the violent movement of large masses of air to form the wave itself. These blast winds have velocities (perpendicular to the plane of the wave) reaching several hundred kilometers per hour. They last only a few seconds but can produce considerable damage through drag forces and by the production of large numbers of low-velocity

secondary missiles, the size and nature of which depend on the environment. A high percentage of blast trauma will be caused by such missiles and a large number of patients will have multiple missile injuries. Many Japanese at Hiroshima and Nagasaki had dozens of superficial wounds caused by flying glass and debris. These types of injuries will vary greatly in severity but, in general, there will be a relatively low incidence of deeply penetrating injuries. However, when massive numbers of casualties must be quickly sorted and prepared for evacuation and treatment, a significant number will have penetrating wounds which easily may be overlooked until clinical signs become obvious. Otherwise, the nonpenetrating missile injuries will not be severely disabling unless critical parts of the body are involved, such as the head, face, neck, or hands.

Combined injuries, missile plus other mechanical injuries or burn injuries, will be very common, and these other injuries will be more important in determining the management of the patients. Radiation injury, sustained at the time of the missile injuries, will not generally cause signficant complications in healing of minor wounds other than transient delay of incompletely healed wounds and increased risk of infection. This is because the clinical manifestations of the radiation-induced bone marrow depression do not occur earlier than 2 weeks and often not until after 4 to 6 weeks. Minor missile injuries will be healed, or nearly so, by then. Severe injuries and open, incompletely healed wounds will be more seriously affected.

The combined effects of the severe drag forces and direct overpressures on structures are very severe and many people will be trapped in collapsed buildings or heavily damaged vehicles. Crush injuries will be very common, particularly in built-up or heavily forested areas, or among personnel caught in severely damaged vehicles. The crush injuries sustained will be the same as those seen in other situations and their management is basically the same. However, pure crush injuries without other associated injuries will be relatively uncommon. The other injuries include low-velocity, secondary missile injuries, burns, and radiation. The associated morbidity and mortality caused by the combination of injuries will be high, particularly among those patients with burns, and

priority of treatment for patients with crush injuries will be low in sorting.

If patients are exposed to radiation at the time of their crush injuries, radiation sickness will not become clinically evident or significant for several days to a few weeks. Many patients then will be past the critical phase of their crush injury.

There is no adequate information on how a crush injury might modify the severity of the bone marrow depression caused by radiation or, conversely, how the radiation injury might affect the recovery from the crush injury. However, it has been established that bone marrow depression does delay healing of wounds and does result in decreased resistance to infection and increased hemorrhagic tendencies.

Delay in healing of fractures, commonly associated with crush injuries, has been demonstrated experimentally; this effect, at least, should be expected. The delay should be about 2 to 3 weeks in patients with recoverable bone marrow depression.

TRANSLATIONAL INJURIES

The blast wind drag forces will result in violent displacement of man himself. Many casualties will suffer multiple blunt trauma from being thrown against other objects and structures or from being tumbled in displaced vehicles. Head injuries and multiple fractures will predominate, but internal injuries will also occur.

RADIATION INJURIES

The detonation of a nuclear weapon produces large amounts of ionizing radiation in two basic forms: electromagnetic (*gamma*) radiation, which travels at the speed of light and is highly penetrating, and particulate (*alpha, beta,* and *neutron*) radiation. Of the particulate radiations, only the neutron is highly penetrating, whereas the alpha and beta are not. All four types are present at the time of the detonation, but the gamma and neutron are by far the most important. All but the

neutron radiation are present in fallout and, in this instance, the gamma is the most important.

Ionizing radiation is emitted both at the time of the nuclear detonation and for a considerable time afterward. That emitted at the time of the detonation is termed *"prompt radiation"* and is produced by the nuclear reactions of *fission* and *fusion.* The significant part of prompt radiation consists of a mixture of gamma and neutron radiation, most of which is emitted within a few seconds of the onset of the detonation. However, the duration of significant emission may be longer, particularly with larger weapons. One minute has been established as a reasonable time, after which there is no significant amount of prompt radiation, regardless of the type of weapon or circumstances of the detonation.

Residual radiation is that which persists beyond the first minute after detonation. Its source is the variable amount of residual radioactive material produced by a nuclear detonation. The nuclear reaction of fission transforms uranium or plutonium into a large number (about 150) of radioactive isotopes, termed *fission products,* which constitute by far the most important source of residual radiation. In addition, small amounts of unfissioned bomb material, and material in which neutron radiation has induced radioactivity, are present. All of these residually radioactive materials will be found in fallout.

Fission products are the major radiation hazard in fallout since a large number of them emit penetrating gamma radiation and, as a result, can be hazardous even at great distances. They have half-lives varying from fractions of seconds to several years, but most have half-lives in the range of days to weeks. As a result, the total amount of radiation emitted by a typical mixture of fission products is quite intense early and remains hazardous until the activity decays down to negligible levels. This takes several days to several weeks, depending on the original level of activity. However, some isotopes with very long half-lives will be present and detectable for many years.

Figure 8 shows that fallout activity decays down to $\frac{1}{10}$ of its base time value by 7 hours after the time of detonation. H+1 is 1 hour after detonation and is a useful base time with which to compare subsequent fallout decay. By this time, a significant part of the early fallout will have deposited itself close to the

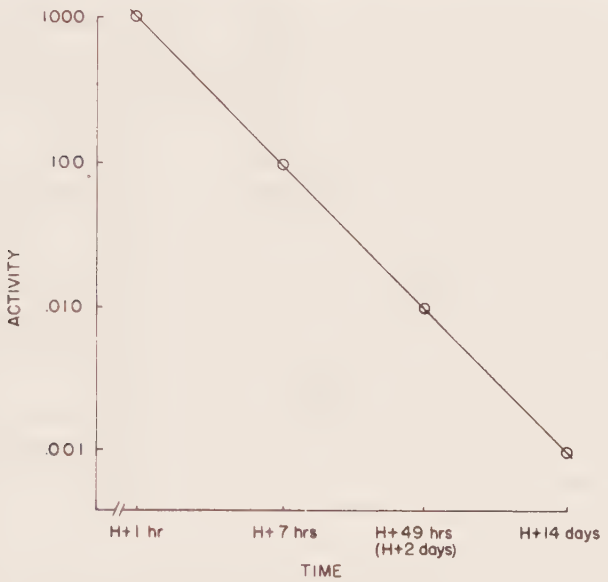


FIGURE 8.—Fallout decay with time after detonation. Fallout activity decreases by a factor of 10 for every sevenfold increase in time after burst. Fallout activity is referenced to 1 hour after burst (H+1 hr), which is used as the base value in fallout calculations.

point of detonation. Deviation from this decay curve will be common, depending on the interplay between the various factors controlling rate of deposition of fallout and the distance involved. At greater distances from the point of detonation, it may take several hours before fallout will begin to deposit and be detectable. A significant amount of radioactive decay will have already occurred while the radioactive material has been airborne and, as a result, the rate of decay, once all the fallout is on the ground, will be similar to the later part of the curve shown in figure 8. If fallout in a given area is a mixture from several detonations at different times, the observed rate of decay may be quite different from this ideal example. Under these circumstances, the rule of thumb that fallout will have decayed to negligible values by 2 weeks may not be applicable. It should be obvious that instruments designed to measure fallout activity must be available and used to evaluate the true hazard.

Not all the uranium or plutonium in a weapon is fissioned,

and fallout, which contains residual weapon material, will contain small amounts of these elements. They add little to the hazards of fallout since they are alpha emitters and are not an external hazard unless ingested or inhaled. They must be incorporated into body tissue to do damage and their relative insolubility greatly minimizes this hazard. Ingestion or inhalation of contaminated material should be avoided.

Neutrons can induce radioactivity by becoming incorporated into the nuclei of the atoms of a wide variety of materials. The resulting unstable radioactive isotopes have very short half-lives and emit beta and gamma radiation. They will include vaporized weapons material and, in many instances, a variety of material found in that small area of terrain which is immediately under the point of detonation (ground zero) and which is subjected to a significant neturon flux. In either situation, the total amount of material involved is small and the contribution of these processes to the total fallout hazard is negligible.

Formation of Fallout

Because of the exceedingly high temperatures generated in a nuclear detonation, all the fission products and the weapon residues are vaporized. As they cool and recondense, they solidify as extremely small particles. In an airburst, these particles will remain suspended in the upper atmosphere (stratosphere) for long periods of time, descending slowly over a period of years and over large parts of the earth's surface. This occurs during the atmospheric testing of weapons. Under such circumstances, there is no significant early or local fallout. When a detonation occurs within a certain critical distance of the surface, however, severe updrafts cause large amounts of terrain debris to be sucked up into the fireball. As a result, as the radioactive materials cool and condense, they become affixed to relatively large particles of dirt and debris. These large particles, with their radioactive contamination attached, tend to fall back to earth rapidly and locally, resulting in high levels of radioactivity downwind from the point of detonation. On occasion, this type of fallout is visible while it descends.

The major hazard in this type of fallout will be external whole body irradiation from gamma-emitting isotopes since they do not actually have to be on a person's skin to cause damage. Gamma radiation has a very long range in air, and large amounts of gamma material scattered uniformly over many square kilometers can produce a high level of penetrating radiation, which is a hazard to anyone occupying or passing through the area, even though he avoids direct contact with the fallout material. Even personnel traveling through in vehicles will be exposed, although vehicles can provide significant reduction of exposure because of the ability of most metals at least partially to scatter or absorb gamma radiation. The dose rate inside a tank, for example, may be only 4 to 10 percent that outside.

The potential injury incurred is a function of the amount of time spent in the fallout field as well as the dose rate present, since these factors together determine the total dose absorbed.

The beta-emitting isotopes in fallout are not a hazard, unless a person is directly contaminated with or ingests them. External contamination can result in a moderate degree of skin damage somewhat similar to a thermal burn, and incorporation into body tissues can result in organ damage of long term significance. These latter effects—that is, interference with specific organ function, carcinogenesis, and accelerated aging changes—are not manifested for months or years, and acute whole body irradiation, with resulting radiation sickness, will not occur. Therefore, in combat situations, the beta-emitting components of fallout are not considered to be a serious hazard.

Whole Body Irradiation

As stated previously, the primary hazard of nuclear weapons radiation, whether prompt or residual, is whole body irradiation. Prompt radiation (gamma and neutron), at the time of the detonation, will be associated with blast and thermal effects and the clinical effects of radiation injury will not be evident at the time of hospitalization for the other injuries. The clinical phase of radiation sickness, which develops after a latent period, typically will result in serious complications of the other injuries; that is, delayed healing, hemorrhage, and increased

risk of infection. Radiation sickness by itself will occur but will not be a common result of single prompt exposures.

Irradiation, as a result of fallout, may or may not be associated with other injuries and, in many instances, uncomplicated radiation sickness will occur following either single or multiple fallout exposures. In other instances, the radiation injury or sickness will precede or follow other combat injuries, resulting in a great variety and complexity of interaction between the various kinds of injuries.

Radiation sickness caused by whole body irradiation may be lethal within a few days to several weeks, depending upon the dose sustained. Clinically, radiation sickness occurs in a dose-dependent pattern of three syndromes, determined by the organ system most seriously involved. These are: the *central nervous system syndrome,* caused by very high doses and uniformly fatal within 2 to 4 days; the *gastrointestinal syndrome,* due to somewhat lower doses but also uniformly fatal; and the *hematopoietic syndrome,* caused by still lower doses and associated with the possibility of recovery and survival.

The central nervous system syndrome will be extremely rare in combat. The gastrointestinal syndrome will be relatively uncommon but may be seen. The hematopoietic syndrome will be the most commonly seen.

All three syndromes have certain characteristics in common. These include:

1. An initial nonspecific response.
2. A latent period.
3. A clinical phase.

Initial response.—Within a few hours after a prompt exposure, all patients, regardless of which syndrome later develops, pass through a nonspecific, transient period of malaise, weakness, anorexia, vomiting, and diarrhea. This response is probably toxic in nature due to tissue breakdown products associated with radiation-induced cellular damage. The exact mechanism responsible or the cell mass involved is not known. This initial response or irradiation lasts up to a few hours and then subsides. It is followed by a latent period during which there are no significant symptoms or obvious physical signs of radiation injury. At present, no diagnostic clues are available to establish firmly the presence or extent of radiation injury

during the initial response phase. Its severity and duration are not reliable indexes of the degree of radiation exposure and it may be absent following low dose-rate fallout exposures despite their magnitude.

A number of patients who are evacuated to hospitals for other injuries may manifest this initial response just before or during the first few hours of hospitalization. Since it is a nonspecific response and cannot be used to predict the degree of radiation injury or prognosis, its presence should not be used as a criterion for selecting patients for treatment. In addition, its signs and symptoms easily could be masked by other severe injuries. Therefore, its absence cannot be used as evidence that there has been no radiation exposure. This will be particularly true following fallout exposures since this response is not so likely to occur when the exposures are to low dose-rate radiation.

Latent phase.—All three syndromes have latent periods between the initial response and the onset of the clinical phase. This latent period is shortest for the central nervous system syndrome, lasting a few hours to 3 days, and longest for the hematopoietic syndrome, 2 to 6 weeks, with an occasional patient demonstrating an even longer latent period. The gastrointestinal syndrome has an intermediate latent period of a few days. This phase is characterized by a feeling of relative well-being.

Occasional patients will demonstrate acute loss of hair during the second week after exposure and this may be a good clue that significant radiation exposure has occurred. This may precede the clinical phase and should warrant medical evaluation, even if other clinical signs and symptoms have been absent. Hematologic studies will show evidence of bone marrow depression before clinical symptoms develop. A peripheral blood count will show white blood cell depression, particularly of the lymphocytes (fig. 9).

Clinical phase.—The clinical phase follows the latent period and many patients will not be hospitalized until this time unless they have had other injuries for which they required treatment. As noted previously, there are three distinct syndromes, depending upon the dose of radiation sustained, as follows:

1. Central nervous system syndrome. The dose of radiation

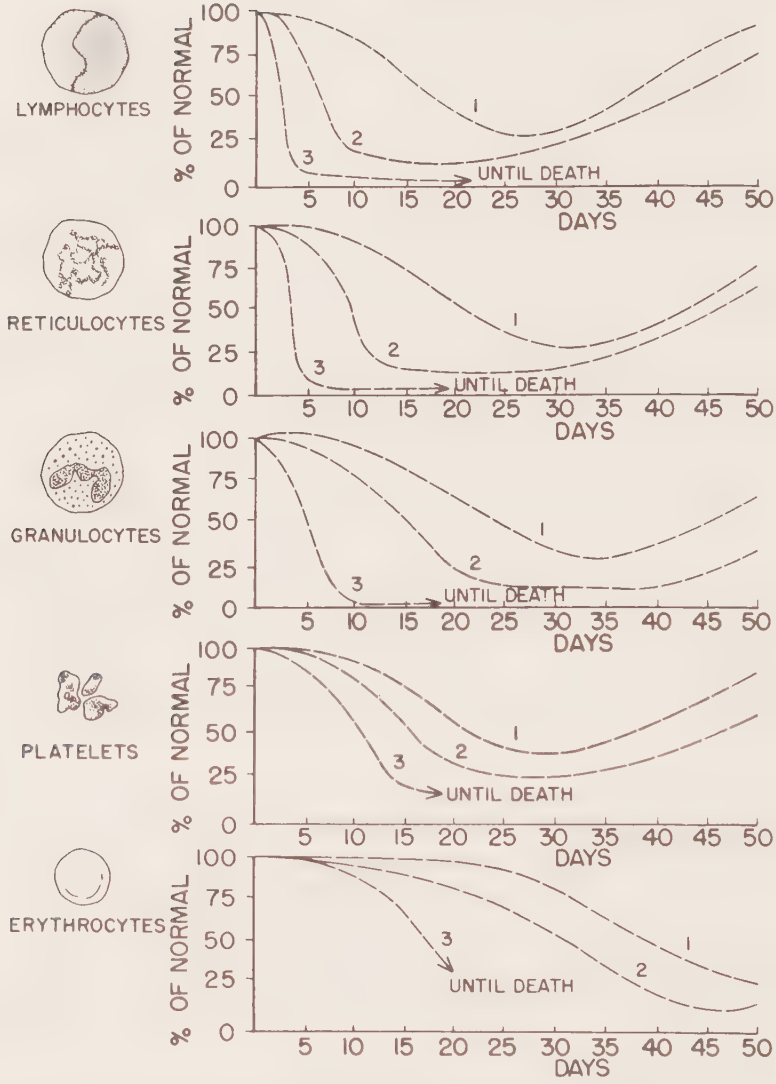


FIGURE 9.—Typical blood cell response to whole blood irradiation. The curves marked 1 represent the response to a low dose radiation, which has a low probability of lethality but which requires hospitalization. The curves marked 2 represent a typical response to a midlethal dose of radiation. The curves marked 3 represent a response to a dose at or above an LD 95–100 level, the point at which the gastrointestinal syndrome typically starts.

required to cause each type of clinical response varies considerably. The central nervous system syndrome requires very high doses, 3,000 rads or more. Such doses are almost impossible in a battlefield situation except for unprotected personnel exposed to extremely intense fallout very close to the point of surface detonation of a large weapon. Therefore, these patients will be extremely rare and most cases will never survive to be seen in medical facilities because of other lethal injuries.

The clinical course of the central nervous system syndrome is one of progressive depression leading to coma and, finally, death. Early, patients will be ataxic and convulsions are frequent as the clinical condition deteriorates. This syndrome progresses too rapidly for significant hematologic changes to occur; therefore, diagnosis will not be easy, particularly if patients have sustained head injuries.

2. Gastrointestinal syndrome. The gastrointestinal syndrome is caused by doses in the range of about 1,000 rads and higher. These doses will not be common, but exposure to prompt radiation from small weapons or to intense levels of fallout will result in a small number of such patients. Small numbers of patients with this type of radiation sickness were seen among the victims at Hiroshima and Nagasaki.

A typical patient with this syndrome will have been hospitalized for other injuries and will, within 4 to 5 days of injury, develop severe, bloody diarrhea. A peripheral blood count will show depression of lymphocytes and beginning depression of other leukocytes and platelets. Differentiation from an infectious, nonradiation-induced diarrhea, superimposed upon radiation-induced bone marrow depression, could well be difficult because of the widespread occurrence of the various dysenteries in combat. As the bone marrow depression becomes more severe, a point will be reached from which recovery will be impossible. Such patients eventually will succumb to the effects of overwhelming infection and hemorrhage, despite massive fluid, electrolyte, and blood replacement and antibiotic therapy. If patients with gastrointestinal damage are not treated, they will die early due to their massive fluid and blood losses. Replacement therapy can prevent this type of death, but, then, such patients will progress to the clinical phase of irreparable bone marrow injury. The survival time of such

patients will vary but may be a few weeks. They could constitute a severe burden on a hospital facility.

3. Hematopoietic syndrome. Patients with small exposures who do not develop the gastrointestinal syndrome will have longer latent periods before the clinical picture of bone marrow depression becomes evident. This may take from less than 2 weeks to more than 6 weeks to develop, but, in most cases, the latent period will be from 2 to 3 weeks.

The degree of bone marrow depression will vary with the dose of radiation sustained, and the probability of survival is directly related to the probability of recovery of the bone marrow. Figure 9 shows different patterns of response of the peripheral blood count to different doses of radiation.

The clinical picture presented by patients with bone marrow depression will vary, depending upon the presence and nature of other injuries. In uncomplicated radiation sickness, the clinical picture will reflect the increased bleeding tendencies which develop. These patients will develop extensive hemorrhages throughout their bodies. Subcutaneous petechiae and ecchymoses and extensive gastrointestinal bleeding will be common. Decreased resistance to infection will accompany the hemorrhagic diathesis and infection will be the primary cause of death. Treatment will be limited to supportive measures, transfusion (preferably with fresh whole blood), and antibiotics. Bone marrow transplantation is not practical therapy in the field.

This syndrome is associated with a chance for survival, depending upon the ability of the bone marrow to recover. Bone marrow recovery and associated favorable prognosis can be determined by serial peripheral blood counts.

Combined injuries.—As noted, radiation injury will be associated with other injuries in a large number of patients. The radiation exposure may occur simultaneously with the other injuries, in which event the clinical phase of radiation sickness will come sometime during the course of recovery from the other injuries. With supralethal doses of radiation resulting in the gastrointestinal syndrome, the primary clinical picture of the syndrome will predominate and any lesser effects from radiation on other injuries will be secondary. Following lower doses, however, the bone marrow depression will have significant

effects upon the clinical course of certain types of wounds and injuries.

Closed wounds will not be affected greatly, but open wounds, particularly burns, will demonstrate delays in healing. Granulation tissue will disappear and the wounds will become pale. In addition, they will bleed quite easily. Wound infection, caused by both exogenous and endogenous organisms, will become a severe problem. Closed simple fractures will not be markedly affected although some delay in union may occur. Open fractures or severe fractures in which infection is a probable complication are dangerous.

Organisms which are not normally pathogenic may become a serious threat to these patients and prophylactic antibiotics are required. The prognosis is related to the probability of recovery of the bone marrow. If the patient survives long enough for recovery to occur, normal or near normal healing of wounds is possible.

If radiation exposures are not coincident with other injuries, the interactions become more complex. The timing of the clinical phase of the radiation sickness with the clinical course of the wounds is a critical factor. For example, if bone marrow depression occurs late in the course of recovery from wounds, there will be little or no interaction. On the other hand, if a patient late in the latent period or in the middle of the clinical phase of radiation sickness sustains serious injury or requires extensive surgery, the prognosis will be very poor. Such a patient should not be subjected to surgery unless its necessity balances the risk and the procedures are required to save life.

Dose-Response

Animal experimentation suggests that a variety of injuries and stresses sustained before radiation may significantly alter radiation sensitivity, increasing resistance to radiation. These results cannot easily be translated to man, particularly man subjected to the many and varied stresses of combat. Therefore, it is reasonable to expect a wide variation in the dose-response relationship in combat. The factors causing this include those related to variations in the nature of the radiation and the

conditions of exposure and those related to variations in the physiological state of the individual.

Physical factors.—Physical factors are as follows:

1. Dose rate. Variation in the rate at which radiation exposures occur has a profound effect on the dose required to produce a given effect. The difference between the dose rate of prompt radiation at the time of the detonation and the very low rates in low-level fallout can be several orders of magnitude. How large a variation this will cause in the dose-response relationship in man is not known, but it should be significant, at least doubling the dose required for a specific response.

2. Nature of radiation. Prompt radiation is a mixture of gamma photons and neutrons. Neutrons are more damaging than are gamma photons and prompt radiation will cause more damage than if it is made up of gamma photons alone. This factor will accentuate the variation due to the differences from dose rate described.

Physiological factors.—Physiological factors are as follows:

1. Recovery. If doses of radiation are small and intermittent, some degree of recovery between doses will occur. The rate or amount of recovery varies considerably with the length of time between doses and the species involved. Quantification for man under battlefield conditions, therefore, is not possible. The degree of recovery will be a significant factor in the response expected from intermittent exposures to very low fallout levels. Many people may be expected, therefore, to accumulate several hundred rads without evidence of acute illness. They will, of course, run a serious risk of late effects, particularly carcinogenesis and accelerated aging.

2. Partial body irradiation. Damage to critical tissue may be avoided if the whole body has not been uniformly irradiated. The bone marrow, in particular, is a tissue that occurs throughout the body and, if a significant fraction of bone marrow, even dormant, is not injured, recovery is much more likely and rapid. Man is a large target, and nonuniform irradiation will occur in a significant number of exposed individuals in combat.

3. Coexistence with other injuries. Particularly as a result of exposures to fallout, there will be considerable variation in the pattern of combination of radiation injuries with other injuries. The resulting types and degrees of interaction will

be highly variable, depending upon the severity of the injury and the dose of radiation.

As noted previously, studies in animals have indicated that significant stresses caused by a wide variety of injuries and physiological stresses before radiation may result in marked increases in resistance to radiation; that is, doubling of the dose required to obtain a 50-percent mortality rate. Injuries following radiation have the opposite effect: profoundly increasing morbidity and mortality. The latter response is easily explained in terms of increased risk of infection and hemorrhagic complications of injuries, whereas the former is probably a function of a stress-induced adrenocortical response.

Dosimetry

In combat, there will be a marked variation in the dose associated with a given effect. As a result, exposure data, such as those obtained from personal dosimeters or estimates based upon area monitoring, are of extremely limited value as aids to treatment decisions in individual patients. This applies as well to the problem of sorting patients in mass casualty situations with present field dosimetry techniques. At the present time, since there has been no field combat experience with nuclear weapons, radiation exposure data should not be used as sorting criteria. The other injuries patients present and the total clinical picture should be evaluated, as described elsewhere in this handbook, and these should be used as the primary criteria for selecting patients for evacuation and treatment.

CHAPTER VIII

Multiple Injuries

Casualties having multiple injuries are difficult to treat because of synergistic effects of the pathophysiologic disturbances of multiple organ systems, increased frequency and severity of shock, and competing priorities for immediate care of the various injuries. Wounds involving more than one organ are characteristically more frequently lethal.

ETIOLOGIC CONSIDERATIONS

The patients in this group most often have sustained multiple missile wounds involving a number of organs or multiple anatomic areas. Patients also are frequently received with multiple injuries caused by missiles but complicated by trauma of other kinds, as follows:

1. Thermal trauma (burns or cold injury).
2. Physical trauma, including blast injury, underwater compression as seen in submarine or ship crews, injuries following decompression (aviation or diving crews), crushing injury, electrical injury, and rapid deceleration injuries as commonly seen in aircraft and vehicle accidents.
3. Chemical trauma such as phosphorus burns, exposure to organic fuels or propellants; injuries resulting from other chemical agents causing either cutaneous, respiratory, or other systemic irritation, or depression of the nervous system.
4. Ionizing radiation injury with either local or total systemic effects.

These special injuries are seen with increasing frequency either in combination with the usual battle wounds or in combination with each other. Men working in various military specialties are subject to combinations of injuries which may be unique to their occupation and environment. Physiologic disturbances secondary to multiple factors such as climatic or environmental temperature extremes, dietary inadequacies, acute or chronic infectious diseases, and systemic poisoning also must be considered and corrected.

MANAGEMENT

Diagnosis, Triage, and Evacuation at the Division Level

A thorough examination is carried out at the battalion or division medical facility initially receiving the patient. Additional problems to be countered are those imposed by the tactical or military situation and often the generally unfavorable condition of the patient; that is, dehydrated, grimy, clothed, burdened with equipment, and frequently confused by the wounding blast or sedation given in the field. Under these circumstances, an accurate medical history is difficult to obtain. Unless great care is taken, it is easy to overlook one or more injuries.

The best way to avoid a serious oversight is to remove the patient's clothing completely and consider systematically all the injuries which could result from a particular type and trajectory of missile or from other trauma. A thoracoabdominal blast injury, for example, should be considered following a mine explosion. Carbon monoxide poisoning and burns of the respiratory tract must be considered in casualties burned about the face or in a fire in a closed environment (such as tank or armored personnel carrier). Shock is frequently present, is often of severe degree, and is usually proportionate to the number and magnitude of injuries sustained.

After examination and identification of injuries, the degree of urgency and priorities of treatment must be established. Immediately, life-threatening problems must be corrected; general

first aid measures, establishment of the airway, control of hemorrhage, and initiation of resuscitation are carried out according to usual routines (see ch. XIV). The patient with multiple injuries presents special problems for consideration during evacuation. If these patients are to survive, essential care must continue en route to a definitive treatment center. Skilled medical attendants are needed to maintain the airway, support the respiration, control hemorrhage, and insure the adequacy of blood or fluid volume replacement. Rapid helicopter evacuation alone is not a substitute for adherence to the above principles, nor does it permit one to ignore the need for adequate fracture immobilization. Concise, accurate records of the injury, of the types of wounds, and of the treatment administered are mandatory to facilitate subsequent appropriate medical care.

Preparation for Initial Surgery

Patients who are seen by medical personnel for the first time at a hospital are evaluated carefully in the manner already described. The accuracy of findings and response to previous treatment are reassessed in all admissions along the evacuation chain. Priorities for care of various wounds in the same patient must be established. While many patients with multiple extensive wounds can be treated successfully, the potential lethality of certain wounds—that is, a massive central nervous system injury or a 90-percent third-degree burn—must be assessed in determining a lower priority for treatment.

Although an oral airway may be adequate in some patients, an endotracheal tube will be mandatory in others to assure an adequate airway. Where indicated, chest tubes are inserted and connected to closed drainage (see pp. 308–309). Major bleeding must be controlled and blood volume replenished, preferably with type-specific blood. Intravenous routes are established with due regard for the site of major injury; for example, a major abdominal injury is best managed with large gage cannulae placed in the upper extremities or neck. Other measures important in a patient with multiple injuries are evacuation of the stomach by nasogastric suction and the insertion of

an indwelling catheter to measure the urinary output and determine the presence or absence of hematuria. Unstable fractures must be splinted either by conventional means or with the radiolucent inflatable splint before further transport. These splints are ill-suited for fractures of the femur or humerus. Inflatable splints also reduce blood loss. However, they may present a threat to the circulation if inflated other than by mouth and at the time of air evacuation due to the expansion of air in the splint at flight altitude.

Appropriate roentgenograms must be obtained and should include special studies when indicated, such as intravenous pyelograms or cystograms in abdominal and pelvic wounds. Abdominal injuries which are produced by missiles entering through the thigh, buttock, or back are easily overlooked. Abdominal radiographic study in such wounds is particularly indicated.

The lack of response to vigorous resuscitation may indicate immediate surgical intervention with control of major internal blood loss. However, other cases which produce or simulate shock must be considered; for example, drug overdose or other poisoning, cardiac tamponade, cerebral malaria, and other infectious diseases. The complication of cardiac arrest usually is treated by closed cardiac compression; however, open cardiac compression may be required.

Operative Management

The order of priority of wound care is often difficult to establish. In general, those injuries most life-threatening are treated initially; thereafter, good judgment must prevail. For example, a patient with both thoracic and abdominal injuries should have definitive operative correction of a lacerated bronchus before a repair of multiple intestinal injuries. Definitive care of intracranial, facial, ocular, and hand injuries frequently must be delayed until other more severe injuries have been cared for. Usually, initial operative care of major chest, abdominal, or extremity wounds is performed at a forward hospital. After stabilization, the patient can be transferred to a larger supporting hospital for the appropriate care

of remaining injuries. Such staging of procedures, even though requiring a second anesthetic, is safer than a long evacuation in the presence of a bleeding major wound. Following lengthy operative procedures, it may be possible to delay other procedures, such as minor wound debridement, until the patient is stable.

Surgical policy should provide sufficient personnel to insure appropriate care while reducing operating room and anesthesia time to a minimum. When the situation permits, this may best be accomplished by having separate teams operating on different regional injuries simultaneously. If the wounds are unrelated, it may be necessary to operate on various anatomical areas in successive procedures. Where possible, for example, a buttocks wound should be debrided and bleeding controlled before exploring the abdomen. Patients in shock with continued blood loss are extremely unstable after lengthy operative procedures, and cardiac arrest is likely to occur if the procedures are performed in reverse order.

The simplest, lifesaving surgical procedure consistent with established principles of combat surgery is all that should be attempted at this time. Unnecessary or meddlesome procedures, such as resection of undiseased appendixes or a Meckel's diverticulum during laparotomy and bowel repair, impose an unacceptable added risk to the patient.

Special Considerations

Despite optimal attention and treatment by personnel at all echelons of care, the patients in the multiple injury category are at extremely high risk. Respiratory support with automatic ventilators is frequently the only way to counteract the pulmonary insufficiency and fatigue factor common to this group. This is particularly true in patients who have major blast injuries, hepatic wounds with concomitant pulmonary contusion, and thoracoabdominal wounds; patients who have required cardiorespiratory resuscitation; and patients with severe sepsis.

A policy of restraint in intravenous crystalloid fluid administration during resuscitation and operation will continue to be

effective, and the likelihood of posttraumatic pulmonary insufficiency is lessened. This policy does not preclude the administration of large volumes of blood or colloid when indicated.

Constant vigilance and an inquiring attitude will help to define confusing problems and provide solutions to seemingly impossible problems.

PART II

Response of the Body to Wounding

CHAPTER IX

Shock and Resuscitation

Shock may be defined as a clinical state in which inadequate capillary perfusion is the common denominator. There is a slowing or cessation of capillary blood flow, particularly in the viscera. This can be caused by a wide variety of medical and surgical conditions. Arterial blood pressure, cardiac output, arterial blood flow, and blood volume, although important in determining capillary flow, are not the only factors having an influence; and shock may be present, therefore, when all of these parameters are normal or above. With the exception of some cases of central nervous system trauma and drug or anesthesia administration, shock is always associated with arteriolar vasoconstriction brought about by high catecholamine levels. In fact, the signs of shock—rapid, thready pulse; pale, sweaty skin—are the results of a high catecholamine level. Function of all organs decreases, most notably that of the kidneys, liver, heart, and brain. With appropriate treatment, complete recovery is possible even after many hours. It is a mistake to regard a wounded man as being in "irreversible shock" merely because he has been severely injured, or because many hours have passed.

PATHOGENESIS AND ETIOLOGIC FACTORS

Shock is the result of pathophysiologic imbalance between the stresses of injury and the defense mechanisms of the body. These stresses are of three kinds:

1. Those acting directly on the central nervous system (neurogenic shock).
2. Those causing fluid losses (oligemic shock).
3. Those resulting from systemic toxicity (septic shock).

There are three major defense mechanisms:

1. The secretion of adrenal hormones, which can produce vasoconstriction, cardiac acceleration, and splenic contraction.
2. The secretion of antidiuretic hormone which facilitates maintenance of optimal blood volume.
3. An increase in available carbohydrates from rapid glycogen mobilization through hypothalamic stimulation.

There are three main pathophysiologic changes associated with shock:

1. Progressive loss of tissue perfusion leading to anoxia of vital cells and ultimately terminating in death. A fall in cardiac output resulting from blood loss may cause or contribute to this decrease in tissue perfusion.
2. Early syncope from decreased cerebral perfusion.
3. Sustained hypotension which may lead to hepatic and renal failure despite recovery from shock.

PREDISPOSING AND AGGRAVATING FACTORS

Circulatory collapse may be hastened or aggravated by a number of factors, including nervous reactions, such as fear, fatigue, and pain; failure to immobilize injured extremities; movement of the injured parts; sudden shifting of the position; traumatic handling during evacuation; fluid and electrolyte imbalance as the result of vomiting, diarrhea, and excessive sweating; a hot environment; overdosages of morphine and other drugs such as Thorazine and muscle relaxants; surgery under inadequate anesthesia or analgesia; prolonged and traumatic surgery, especially surgery in which traction on the mesentery is required; and massive contamination of the peritoneum.

CLINICAL MANIFESTATIONS

Neurogenic Shock

Neurogenic shock is due to stimulation of the autonomic nervous system, which results in either widespread vasodilatation or the inhibition of vasoconstriction. After injury, or after an emotional upset, the blood is shunted from active circulation to the voluntary muscular system and the gastrointestinal system, thus depleting other vital systems, including the brain. As a result of this shift, hypotension occurs rapidly.

Neurogenic shock may occur in the early stage after wounding with or without significant blood loss from the manipulation of fractures or from other painful or unpleasant procedures. It also may occur in some head injuries, presumably as the result of disturbance of the medullary centers, and in some spinal injuries because of paralysis of the vasomotor fibers in the thoracolumbar outflow tracts.

Neurogenic shock appears promptly after the precipitating incident. Some persons are more susceptible than others. Symptoms and signs include pallor, deep sighing respiration, vomiting, syncope, bradycardia of 60 or less, and hypotension. When neurogenic shock is associated with head injuries, the pulse is slow and may be irregular, and the respiration is irregular and often stertorous.

Oligemic Shock

Oligemic shock is characterized by loss of circulating fluid volume. It can be classified, according to its main causes:

1. Direct loss of blood, either external or internal.
2. Loss of blood or plasma by seepage, as in crushing injuries, massive muscle damage, intestinal infarction, burns, and contusions. In these injuries, the loss of fluid is extracirculatory; blood and plasma leak from the vessels, capillaries, and lymphatics and are sequestered in extravascular spaces.
3. Loss of intestinal fluids, as in severe vomiting or diarrhea, ileus, intestinal obstruction, and enteric fistulas.

Oligemic shock often merges with neurogenic shock, developing more slowly unless precipitated by massive hemorrhage. When the injury is minor and blood loss is correspondingly small, the complete shock syndrome may not occur; pallor and lightheadedness may be the only manifestations. Minor injuries with some manifestations of shock, such as hypotension and tachycardia, should lead to the suspicion of an undetected injury or concealed hemorrhage. Severe injuries may cause the complete shock syndrome, manifested by faintness, pallor, increasing thirst, dryness of the mouth, coldness of the extremities, and restlessness. The pulse rate increases, and the pulse pressure and volume decrease. The blood pressure, which at first may be sustained or even elevated, may fall suddenly with shifts in the position of the patient. Altered cerebral activity, which is the result of cerebral anoxia, may be manifested either as apprehensive alertness or as apathy.

As vasospasm occurs, the peripheral veins collapse. Because of the decreased blood flow in the capillaries, pallor of the lips and skin increases, followed eventually by cyanosis. A gray mottling of the skin is characteristic. Because of capillary stasis, color returns slowly to the ear lobes or nail beds after the release of digital pressure. Awareness of cold is increased, and often sensibility to pain is decreased. The limbs are hypotonic and often cold and clammy. The urinary output is diminished and the patient may become anuric. Coma is not a usual manifestation of oligemic shock; when it is present, one should suspect an associated head injury.

If the patient's status continues to deteriorate, respirations become rapid, irregular, and gasping. Respirations of this type, hypotension, and evidence of capillary stasis are late indications of fully developed oligemic shock and impending death. Treatment should never be deferred until these signs and symptoms appear, and should never be withheld because of indications of "irreversible" shock.

Septic Shock

Septic shock may result from the following causes:

1. Infection of injured tissues, especially muscle tissue.
2. Absorption of bacterial toxins or toxic products from

damaged tissues when debridement has not been performed or has been performed inadequately.

3. Massive infection of serous cavities, especially the peritoneal cavity.

ORGANIZATION OF A TRIAGE AND RESUSCITATION FACILITY

Prompt, preoperative resuscitation can save many lives. Careful preliminary preparations must be made and resuscitative measures instituted with the least possible delay once a casualty has been received in the resuscitation facility. The triage and resuscitation of casualties in shock require considerable clinical experience and acumen. An experienced medical officer, assisted by an experienced and well-trained staff, therefore, should be in charge of a triage and resuscitation facility. There must be coordination among the triage officer, resuscitation personnel, and operating room personnel. The surgeon must be intimately involved in the coordination and determination of priorities of treatment.

Physical Setting

1. The facility should be a large, well-lighted expanse of uninterrupted space, allowing free movement of people and an unobstructed view of the entire room. Partitions or unnecessary structures which interfere with communication have no place. The triage officer must be able to see the whole situation at a glance from one place to direct the activities effectively.

2. Such a facility should be capable of handling a large number of casualties. The location of this space is important in relation to the transportation which delivers the casualties and to the other services needed for their care. It should be immediately adjacent to the ambulance unloading area or the helicopter pad so that transfer into and out of secondary vehicles is not required, and should be situated close to the operating room. Mobile X-ray apparatus should be close at hand. These arrangements reduce the necessity for moving the patient, which is always deleterious in shock.

3. Supplies and equipment should be immediately visible and accessible without obstructing floorspace. A large number of open shelves lining the walls circumferentially about the triage area will be valuable for this purpose.

4. The blood bank and X-ray facility should adjoin the triage area. Laboratory tests other than cross-matching of blood and determination of arterial blood gases are not needed for initial resuscitation and can be set up in a laboratory closer to the wards and intensive care unit.

5. The facility should be arranged so that patients can be moved easily and rapidly from the triage area or X-ray facility to the preoperative area and the operating rooms. After initial treatment and evaluation, patients should be separated according to priorities. Those who will need major surgery should be moved to a location for further preparation. Those needing only debridement of minor wounds under local anesthesia can be cared for in a separate area.

Equipment and Supplies

1. Frames upon which the stretchers will be placed should always be in position, carefully arranged to allow enough space between patients for easy movement. A minimum of other furnishings is necessary. Aside from a desk or countertop work space for recordkeeping, there should be no chairs or furniture about the working area. Stethoscopes, sphygmomanometers, and devices for suspension of I.V. bottles should be at every stretcher position.

2. Sterile prepacked sets for emergency procedures, such as cutdowns, tracheostomies, insertion of chest tubes, and control of bleeding, should be conveniently located. These sets must include all of the instruments, sutures, and fittings needed for the purpose and should be plainly marked.

3. Suction equipment must be immediately available for airway aspiration.

4. Laryngoscopes and endotracheal tubes with inflatable cuffs should be conveniently located in the resuscitation area. Insertion of an endotracheal tube is a rapid means of assuring upper airway integrity and facilitates the performance of a

tracheostomy. Oropharyngeal airways prevent the tongue from obstructing the oropharynx in the unconscious patient. A ventilating bag with mask and endotracheal tube fittings for manual ventilation should be available at numerous locations.

5. Large bandage scissors must be in each corpsman's pocket and at numerous other places for quickly removing clothing from casualties.

6. Intravenous fluids are needed in large quantities and should be immediately available in the triage area. One bottle of the preferred fluid with tubing and airway inserted should hang in place over each set of litter frames. A blood filtration set should be used to permit subsequent administration of blood.

7. Percutaneous venous catheters are preferable to needles in administering intravenous fluids. The intravenous pathway should be at least 18 gage.

8. Large-bore catheters for chest drainage and sterile tubing for institution of underwater drainage or suction should be available. Heimlich one-way valves attached to chest tubing are acceptable only for temporary purposes.

9. Quantities of prepackaged sterile dressings in various sizes should be in ample supply at every stretcher.

10. Prepackaged sterile syringes are needed in 5-, 10-, and 20-ml sizes. In addition, preheparinized 5-ml syringes will be needed for samples for blood gas determination.

11. Sterile prepackaged sets of urinary catheters will be needed and should be available. Only large balloon Foley catheters should be used.

TREATMENT

The approach to the patient should be directed to the concern for adequate airway, control of major bleeding, and restoration of blood volume. While much of the activity will be carried out simultaneously, a clear idea of priorities is necessary.

Intravenous fluid administration is the single most important factor in the treatment of any type of shock. The two vital questions in proper fluid administration are (1) how much fluid and (2) what kind of fluid.

The amount of fluid required is a much more complicated problem than is at first apparent. It cannot be dismissed as merely replacing lost volume. Not only is it usually impossible to estimate or measure lost volume, but the total blood volume required in shock is often more than a normal blood volume.

The most important method of determining the volume requirements in shock and burns is the accurate determination of central venous pressure. This is the pressure in the right atrium or great veins of the chest. It is not a measurement of blood volume. It is not even a measurement of volume requirements. It merely indicates whether the heart is capable of pumping additional blood volume, and if adequate blood volume is being presented to the heart for its pumping action. There are a number of methods of inserting a central venous catheter. It may be inserted into almost any peripheral vein. The important thing is that the tip of the catheter must be placed within an intrathoracic vein (but not in the right ventricle), preferably in or near the right atrium. Leg veins are less desirable because of the length of the catheter needed and the increased danger of infection. Arm veins are better, but are usually collapsed in shock and may be difficult to fill. Jugular veins are satisfactory. Probably the best vein is the subclavian. It is large, always present in a constant position, and easily dilated by a slight Trendelenburg position. It may be approached either from below or above the clavicle. Probably the supraclavicular approach is the easiest and is productive of the fewest complications. It has proven to be very satisfactory for routine use, however, with some complications; for example, pneumothorax, hemothorax, and hematoma. The percutaneous venous catheter is recommended for convenience. The 14-gage 2-inch needle with the 18-gage radiopaque tubing is preferred. The 12-inch variety most frequently reaches the right atrium.

The patient's neck is first prepared with antiseptic solution. A point 1.0 cm posterior to the junction of the lateral border of the sternocleidomastoid muscle and the clavicle is infiltrated with a local anesthetic solution. With gentle aspiration, the needle is advanced at an angle of no more than 45 degrees to the horizontal plane between the clavicle and the sternocleidomastoid muscle (fig. 10). The approach is from above the

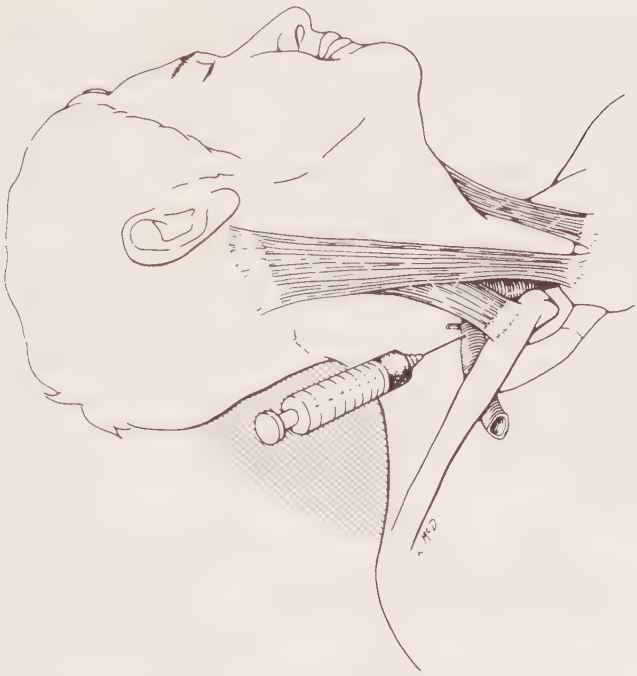


FIGURE 10.—First step in insertion of subclavian catheter. The needle is inserted, roughly bisecting the angle between the clavicle and the sternocleidomastoid muscle, at an angle of no more than 45 degrees to the horizontal plane.

clavicle, with the patient's head tilted slightly away from the side of the procedure, and the feet of the patient elevated 6 to 8 inches, to prevent air embolus and to distend the subclavian veins. When the vein is punctured, blood is freely aspirated and the plastic tubing, still within its sheath, is inserted and threaded down, leaving about 3 inches of catheter on the skin. If subclavian catheterization is unsuccessful, femoral vein catheterization may be utilized.

It is important in preventing catheter emboli that the needle barrel and the polyethylene tubing be fastened with tape. The catheter is connected to a three-way stopcock. One arm goes to a water manometer and the other to a bottle of intravenous fluid. Fluid is administered until it is desired to measure central venous pressure. Then, the stopcock is turned to

divert fluid into the manometer. The stopcock then is turned to connect the catheter with the manometer. The level in the manometer then falls to the central venous pressure. The tip of the catheter must be in an intrathoracic vein, preferably in or near the right atrium. If the catheter tip is in an extrathoracic vein or right ventricle, readings will be meaningless or, even worse, will give false information.

Taking zero level (level of three-way stopcock) at the patient's midaxillary line, 0–5 cm of water is low, 5–10 cm is normal, 10–15 cm is high normal, and above 15 cm is high and a level at which pulmonary edema may be imminent. The catheter is used to measure central venous pressure, to administer intravenous fluids, and to obtain venous blood. The catheter must be kept open with a slow drip of intravenous solution to which 15 mg of heparin and 10 mg of 1-percent Xylocaine per 1,000 ml are added.

Several points in the proper evaluation of central venous pressure need emphasizing. Vasopressors artifically elevate the central venous pressure. Accurate central venous pressure measurements must be taken after vasopressors have been discontinued. Stopping vasopressors lowers central venous pressure. Additional intravenous fluid then may be given, if indicated.

Twenty-five percent of the patient's normal blood volume may be lost with preservation of a normal systemic blood pressure due to compensatory vasoconstriction. In this situation, a normal central venous pressure is more reliable in determining adequate fluid replacement. This information is particularly important when planning spinal or inhalation anesthesia. If, on the other hand, systemic blood pressure is low and the central venous pressure is normal, it is relatively safe to give more fluid slowly and intermittently up to a level of 15 cm of water. There are exceptions to this, however. When adequate blood volume is obtained and the right atrium is receiving adequate return, the normal right heart pumps this volume into the pulmonary artery. If vasoconstriction in the lungs is still present and has not been relieved by physiologic vasodilation, the pulmonary pressure may rise precipitously and pulmonary edema may result, even in the presence of a normal central venous pressure. It may be only with right heart fail-

ure that the central venous pressure rises. This emphasizes the importance of slow and intermittent volume administration when the central venous pressure is above 6.0 cm of water. Readings above 15 cm of water are too high and pulmonary edema may be produced. Therefore, fluid volume administration should be discontinued. If a vasopressor is being given, it should be stopped. Attempts may be made to improve cardiac action by adequate oxygenation and digitalization. A vasodilator such as isoproterenol will lower the central venous and pulmonary artery pressure; in addition, it also has an inotropic action. However, any inotropic agent such as isoproterenol or digitalis may have a detrimental effect on an anoxic heart, causing tachycardia and arrhythmias, which dictates cautious use. In cases of pulmonary edema caused by overhydration, phlebotomy, rotating tourniquets, or diuretics may be required.

In cases of massive injury with obviously severe blood loss, more than one I.V. route may be necessary. The second one should be easier to place after expansion has been started through the initial pathway.

Blood samples for cross-matching should be obtained immediately upon admission of a casualty with serious injuries. These can be drawn through the needle or catheter used to begin the I.V. infusion if it is placed quickly. If, however, O-negative blood transfusion in amounts greater than 2 liters has been administered in forward areas, this type of blood should be continued, only utilizing type-specific blood after a period of 3 weeks.

The type of fluid for beginning resuscitation is much less important at first than the promptness with which any intravenous fluid can be started. Because it is preferable to use properly cross-matched blood, an electrolyte solution is preferable to start. Ringer's lactate is probably the best solution, although normal saline is adequate. Electrolyte solution is needed to replace not only lost blood volume but also lost extracellular fluid which has been depleted, to bolster the shrunken blood volume. Dextrose solutions are inadequate for this purpose and, in excess, may result in water intoxication. When properly cross-matched blood is available, it should be started immediately. When given in massive amounts into a

central vein, some method of warming the blood should be utilized. The volume of each of these fluids should be determined first by observations of central venous pressure, urinary output, and hematocrit. The use of plasma expanders offers little benefit over that of electrolyte alone, especially if blood is available. Sodium bicarbonate should not be given empirically since low perfusion acidosis due to hyperlactacidemia ordinarily will be rapidly improved by adequate volume administration of any kind. Most early traumatic shock victims have an elevated blood pH, and sodium bicarbonate is contraindicated. It may be used in those rare instances where severe metabolic acidosis is proven and immediate correction is mandatory. The need for any colloid in the fluids used for the first hours of resuscitation other than in the form of whole blood is not established. Dextran not greater than 20 ml per kg body weight per day, albumin, and Plasmanate are used. While their colloid osmotic activity makes them more effective in remaining inside the vascular compartment, such materials are not clearly superior to crystalloid solutions. Crystalloids in greater quantities will perform the same function with perhaps a greater margin of safety in terms of overloading the vascular space. As long as the medical officer keeps in mind the fact that crystalloid solutions will distribute throughout the extracellular space, about two-thirds into the interstitial space and one-third remaining within the vessels, the volumes can be controlled safely.

Estimation of blood loss in combat casualties is likely to be grossly inaccurate. Therefore, the replacement of fluid and blood should not be determined by estimated blood loss, but by response to fluid administration in terms of adequate systemic blood pressure, central venous pressure of 5 cm of water or more, and adequate urinary output.

When needed, morphine or other narcotics should be given intravenously in small doses for better control of analgesia. Consideration should be given to morphine which has been administered subcutaneously or intramuscularly in the field, as these doses remain unabsorbed in a patient in shock. When circulation and perfusion have been restored, the drug is mobilized and produces unwanted degrees of respiratory depression.

Vasopressor drugs such as noradrenalin (norepinephrine) are of value in the treatment of neurogenic shock. Their value in oligemic shock is doubtful and not recommended. They may give rise to a false sense of security, for they may raise the blood pressure even when the circulatory volume has not been adequately restored. Skin ischemia, necrosis, and superficial phlebitis are hazards when noradrenalin and similar drugs are used.

Some patients cannot be adequately resuscitated before operation. Particularly in the case of intra-abdominal and intrathoracic vascular injuries, blood may continue to be lost as rapidly as it is replaced. Although this continuing loss may be inapparent because of internal hemorrhage, the nature of the wounds and the inadequate response to treatment should indicate that surgical intervention is urgently needed. These patients may enter the operating room in shock, requiring little or no anesthesia. When the bleeding is controlled, blood volume restoration should be accomplished before continuing with the operation.

CHAPTER X

Physiologic Responses to Trauma

Major combat wounds initiate physiologic responses which may last for many days or weeks. The magnitude and duration of the responses are roughly proportional to the extent of the injury, the interval between wounding and treatment, and the development of posttraumatic complications.

CARDIOVASCULAR RESPONSE

The most vital posttraumatic changes are the hemodynamic responses that result in the preservation of tissue perfusion by contraction of nonvital intravascular spaces and maintenance of hemodynamic equilibrium. Loss of blood is followed by compensatory vasoconstriction and tachycardia, which permits a reduction in blood volume of 20 to 30 percent while maintaining the blood pressure at nearly normal levels. If hypovolemia persists or rapidly progresses below these levels, hypotension results. Unless such a serious deficiency is corrected promptly, death may ensue. Unsuspected hypovolemia may be recognized by early signs and symptoms, such as rapid, thready pulse and pale, cool skin, before development of hypotension. Anesthetic agents depress the autonomic response and, if administered to a hypovolemic patient, the effects of the original injury may be aggravated and shock may result. Therefore, blood volume

replacement ordinarily should be accomplished before anesthesia.

To maintain tissue perfusion in hypovolemic states caused by blood loss, extracellular fluid from the interstitial space crosses the capillary wall into the intravascular space. This transcapillary refill is responsible for the gradual dilution of the concentration of red cells and is recognized by a falling hematocrit. In an injury of moderate severity in which blood loss is less than 1,000 ml, this response is completed within 24 to 36 hours and usually results in a normal blood volume but a decreased red cell mass. The body can tolerate a reduction in concentration of red blood cells far better than a deficit in total blood volume. If the blood volume is maintained at or near normal, a young man can withstand considerable anemia for a short period of time. Therefore, the patient who is in shock because of blood loss usually responds well to prompt correction of the volume deficit with blood plasma, plasma expanders, or crystalloids. A casualty who has received large amounts of plasma substitutes may remain pale and his cardiac rate may be increased until his anemia is corrected by whole blood or packed red cells. However, correction of this anemic state is not a matter of immediate concern. During the phase of hemorrhagic shock when blood flow to tissues is low and venous return to the heart is decreased, the cardiac output is correspondingly reduced. The increase in heart rate represents an attempt of the body to compensate. If the rate rises higher than 120 to 130 beats per minute, the cardiac output decreases due to decreased diastolic filling. When the heart rate returns toward normal, the stroke volume and cardiac output improve.

HORMONAL RESPONSE

Along with the cardiovascular changes after wounding, an increased pituitary and adrenal hormonal output has a stabilizing effect. The net effect of the hormonal response is to augment the cardiovascular response in restoring and preserving blood volume and tissue perfusion. The hormonal response occurs as a normal reaction to the stress of combat, even in the absence of wounding.

Antidiuretic hormone from the posterior pituitary, by conserving water, and aldosterone from the adrenal cortex, by conserving sodium, provide protective mechanisms for the maintenance of blood volume. The increased secretion of these hormones results in a reduced urine output. Concomitantly, potassium excretion increases. Decreased renal blood flow in hypovolemic states accentuates the reduction of urine output. The resulting small urine volume has a high specific gravity (or osmolality) and a low sodium concentration. Other hormones from the adrenal cortex, for example, hydrocortisone, are secreted in increased amounts. The cortical hormones assist the body in responding to the vasopressor hormones (epinephrine and norepinephrine) from the adrenal medulla. These hormones act to mobilize glucose from glycogen stores to provide a source of ready energy for additional cardiac and respiratory work and to prepare the body for the "fight or flight" response. In most instances, nothing is gained by administering additional cortisone. If, for some reason, the adrenal glands are not functioning, as in adrenal injury or hemorrhage, the patient's circulation will be impossible to support; a fatal outcome is likely unless cortisone is given. When in doubt, a single intravenous dose of 300 mg of hydrocortisone may be justified as a therapeutic test.

WATER AND ELECTROLYTE RESPONSES

The combat casualty who was in good health before wounding seldom demonstrates a metabolic or electrolyte abnormality which will prove fatal during the first 24 hours after injury. Death during this period is usually from the loss of whole blood, and the major therapeutic effort at this time should be directed toward controlling hemorrhage and restoring blood volume.

During the first 24 hours after wounding, the plasma sodium chloride concentration falls, due to an intracellular shift of sodium. During the next 36 to 72 hours, grave electrolyte deficiencies may develop because of loss of water and electrolytes in urine, from skin, and from gastrointestinal tract. Salt deficiency will always accompany water deficiency in a casualty

who has had nothing by mouth and has not had replacement therapy. Therefore, signs of dehydration, such as hypotension and mental confusion, cannot be corrected by water alone. If these signs are present, at least 2 liters of a balanced salt solution, such as Ringer's lactate, should be administered.

Seriously injured persons may present in a dehydrated-desalted state because of inadequate intake and increased fluid and electrolyte loss in the combat situation. The electrolyte and water deficiency is aggravated by delay in receiving treatment.

After correction of electrolyte deficiencies, the remaining fluid requirement caused by insensible loss should be supplied by approximately 2,000 ml of 5-percent dextrose in distilled water daily plus electrolyte replacement on a volume-for-volume basis for other external fluid losses. Protein deficiency ordinarily is not a problem in the acutely traumatized individual; it may become a problem in prolonged and complicated convalescence when the patient is in a catabolic state. Hyperalimentation with intravenous amino acids and hyperosmolar glucose is indicated for these patients when the oral route is not available.

Water Depletion

A loss of pure water may occur in acutely injured persons, usually from the lungs (especially with tracheostomy) and sometimes from the skin (as in burns or in hot, dry climates). In such situations, the urine volume is low and specific gravity is high, reflecting good renal function. If deprivation continues, however, decrease in renal circulation and glomerular filtration may lead to permanent impairment. Here the serum sodium concentration is high, the patient is thirsty, then later febrile, flushed, and hallucinating. This is the typical picture of the castaway on a liferaft. Treatment consists of the administration of pure water by mouth or intravenously as 5-percent dextrose in water. If the serum sodium concentration is 150 meq per liter or higher, the patient will need more than 5 liters per day of 5-percent dextrose in water to accomplish the dual goals of restoring previous losses and compensating for continuing water losses.

Salt Depletion

Primary salt deficiency develops in patients who have excessive sodium losses, as with vomiting, diarrhea, gastrointestinal fistulas, or those whose intake consists only of water. Physical signs of advanced salt depletion consist of a decreased blood pressure; softening, shrinking, and longitudinal wrinkling of the tongue; and loss of elasticity of the skin. In this circumstance, a low serum sodium will reflect sodium deficit which is repaired effectively by the administration of salt-containing solutions. It should be remembered, however, that hyponatremia, per se, may represent excess water intake and is not of necessity an indication of salt depletion. In this situation, the total body sodium may be normal, low, or high in the presence of a decreased serum sodium. If hyponatremia is dilutional rather than depletional as a result of impaired water excretion or excessive water administration, large volumes of sodium-containing solutions may be not only unwarranted but also dangerous. In this instance, water restriction rather than sodium administration is the treatment of choice. If doubt exists as to the exact mechanisms, a cautious trial of hypertonic sodium replacement with close observation is warranted.

PATHOLOGIC RESPONSES TO TRAUMA

Kidney

Urinary output.—The decrease of urinary output which occurs as a physiologic response to wounding is the result of both metabolic and vascular changes. Normally, the urinary output is in excess of 500 ml per 24 hours if the blood pressure is within the normal range and if the urinary flow is not mechanically obstructed. As the systolic blood pressure is lowered by hemorrhage to a level of 60 to 80 mm Hg or even lower, the urinary flow decreases and may progress to oliguria; that is, to a volume of urine less than 20 ml per hour, or less than 400 ml per 24 hours.

Obstruction of a urinary catheter is a particularly likely cause of absence of detectable urine. Most patients, even those

with acute renal failure, excrete 50 ml or more of urine per day; when anuria develops, a mechanical reason for it should be suspected. Frequent causes are obstruction or actual destruction of the urethra or ureters by wounds in the pelvic region, spasm of the urethral sphincter and atony of the bladder. Careful physical examination, catheterization of the urinary bladder, and intravenous pyelography, cystoscopy, or exploration, as indicated, will establish the presence or absence of adequate urinary flow from the kidneys. If a urethral catheter has been inserted, it may be obstructed by mucous plugs or blood clots. These are such obvious causes of oliguria and anuria that, paradoxically, they are sometimes overlooked.

Acute renal insufficiency.—Acute renal insufficiency or acute renal failure indicates sudden and essentially complete failure of the excretory function of the kidneys. This complication, in which the pathologic process is acute tubular necrosis (lower nephron nephrosis), must be suspected if less than 400 ml of urine is excreted in a 24-hour period. It is important to recognize, however, that approximately 15 percent of casualties who develop the syndrome of acute renal failure do not have oliguria but may become uremic nonetheless (high-output renal failure). Although urine volumes may be normal to high, a lack of concentration indicates failure to clear solutes. Failure to recognize this fact and to monitor the patient's fluid administration may result in overhydration and fatal circulatory embarrassment of the nonoliguric as well as the oliguric patient with acute renal failure.

Factors which frequently cause acute renal insufficiency are long periods of hypotension; crushing injuries; burns; hemolytic reactions, most frequently from blood transfusions; drug nephrotoxicity; sepsis; and hypersensitivity phenomena.

At first, the urine is pale and dilute unless blood or hemoglobin is present. If hemolysis has occurred, it is characteristically dark brownish red. Proteinuria may be conspicuous for a day or two. Granular and heme-pigment casts soon appear. The specific gravity falls rapidly, and by the third day, it may be as low as 1.010 and fixed.

The BUN (blood urea nitrogen) level rises rapidly. The rate of increase is closely related to the extent of trauma or of factors which influence the catabolic rate. In a massively

wounded and catabolic patient with renal failure, the BUN may rise as much as 120 mg percent per day. Hyponatremia is a frequent finding and is usually attributed to excessive administration of water rather than to an actual sodium deficit. With the development of metabolic acidosis, the serum bicarbonate falls. Hypocalcemia is frequently present. Anemia and leukocytosis are usually present, even in the absence of infection. Because infection is a leading contributory cause of death in acute renal failure, search for foci of infection is mandatory. Diarrhea, sometimes with bloody stools, may develop if uremia persists. Abdominal distention may be marked. Drowsiness, disorientation, muscular twitchings, and even convulsions may occur. Diastolic hypertension of considerable degree is not unusual. Acute pulmonary edema and congestive heart failure are more likely to develop when hypertension is marked, especially if an excess of fluid has been given. Weight loss and hypoproteinemia, progressing to emaciation, reflect the catabolic state, and dependent edema may occur, even when the fluid allowances are less than conventional. The clinical course may be complicated by extensive and progressive infection, impairment of wound healing, and a distinct tendency to bleed.

Many of the abnormalities observed in acute renal insufficiency are the result of potassium intoxication. Because of catabolism, the potassium ion shifts from its normal intracellular status to extracellular fluid compartments. The process may be more rapid in the presence of necrotic tissue or hematoma formation and should be suspected whenever major injury to muscles is present. In the presence of acidosis and uremia, the plasma potassium levels are abnormally high and the extracellular/intracellular potassium ratio is high. Serious degrees of potassium intoxication can occur on the first day after wounding in casualties who are oliguric. Frequently, physical signs or symptoms are inapparent until death is imminent. Neuromuscular and cardiac changes are manifestations of potassium intoxication. Tendon reflexes are diminished to absent, and complete paralysis may follow. Potassium intoxication causes certain electrocardiographic changes, such as high peaked T waves in the precordial leads, a spread of the QRS complex, depression of the P waves, and a sloping ST segment

in the limb leads. Conduction disturbances can lead to ventricular arrhythmia and death. Fatalities from cardiac arrest secondary to potassium intoxication have been observed as early as the fourth day after wounding.

The usual earliest sign of acute renal failure in a wounded man is the appearance of oliguria with no other obvious cause for a decreased urinary output. Volume expansion, monitored by the central venous pressure, will help in identifying and treating cases of prerenal oliguria. This is accomplished by rapidly administering a test load (500 to 1,000 ml) of intravenous fluid, rapidly followed by a single dose of a diuretic agent. The urine specific gravity is usually 1.010 in the syndrome of acute tubular necrosis, and the urine sodium concentration is relatively high (60 to 100 meq/l). The UUN/BUN (urine to serum urea ratio) is usually less than 10 : 1. Electrocardiographic and chemical determinations may confirm the presence of hyperkalemia.

The clinical manifestations of sepsis, shock, and necrosis of tissue which has not been debrided are quite similar to those of uremia, but the differential diagnosis is seldom difficult because these manifestations appear considerably earlier than in uremia. Even though both oliguria and azotemia may be present in the first few days after wounding, the nausea, vomiting, disorientation, and convulsions which occur at this time are not likely to be of uremic origin.

Since renal insufficiency usually is not diagnosed in its incipiency, treatment during this phase, when the only manifestation is oliguria, is vascular volume expansion using blood and other suitable electrolyte solutions with monitoring of the central venous pressure; adequate debridement of any wounds; a trial of mannitol intravenously injected in a 12.5- to 25-g bolus; and the administration of antibiotics as indicated. The concern of the medical officer in a forward unit should be the correction of potentially reversible renal failure by prompt restoration and preservation of adequate blood volume and urinary flow.

In a temperate climate, the total fluid intake for 24 hours, exclusive of blood, plasma, or plasma expanders, should be 500 ml to cover insensible loss, plus the measured output. The measured output is the total of urinary excretion, vomitus,

diarrhea, fluid removed by gastric suction, and fluid lost from burned surfaces. Allowance must also be made for increased insensible fluid losses. These vary according to climatic conditions and body temperature. In humid tropical regions and in febrile states, these losses may be 2,000 ml per day or more.

Maintenance of the proper relationship of fluid intake to fluid output is important, for increasing the fluid intake will not increase the urinary output in acute renal insufficiency. An excessive intake, in fact, will endanger the patient's life. The responsible medical officer must give his personal attention to the calculations. A careful record must be kept, and nurses and aidmen must be instructed specifically about how to keep it. A warning notice to keep the fluid intake-output chart must be displayed prominently on the patient's bed.

The patient's thirst must not be allowed to influence the volume of intake, and close supervision is necessary to insure that he does not overhydrate himself. A daily weight record should be maintained if practical. An increase in weight implies water retention and, therefore, overhydration. A useful general rule is the maintenance of ½ pound daily weight loss under usual catabolic conditions.

Administration of fluids should be orally if tolerated and feasible. When parenteral administration is required, as it often is, it should be a continuous intravenous infusion at a constant rate. It is technically simple to pass a polyethylene catheter into the superior vena cava via a peripheral vein, and little trouble need be expected if the tube is allowed to remain in situ for no longer than 5 days. This technique minimizes the risk of thrombosis, which would be associated with infusion by a needle or cannula in a peripheral vein for this period of time. It also makes movement of the patient simpler and allows central venous pressure monitoring through the same catheter.

Although wound management is essentially the same as in patients without renal failure, early debridement is even more critical, in that damaged tissue aggravates the effects of renal failure. Hypoxia and respiratory acidosis during anesthesia should be particularly avoided, since they may promote release of intracellular potassium into the plasma.

Caloric intake should be maintained by the use of carbohy-

drates and fats, with the complete elimination of protein-containing foods. Hypertonic glucose can be given effectively through a central venous catheter. Potassium should not be administered to the oliguric patient unless the ion is deficient.

The early use of mannitol as an osmotic diuretic has been mentioned. If diuresis results from a 12.5- to 25-g bolus, a sustaining infusion of 20-percent mannitol may be used to titrate an adequate urine volume. Furosemide and ethacrynic acid have received considerable recent attention and probably one should be used as an initial diuretic. Caution is required in their use, however, as serious adverse reactions have been reported, including deafness and death. The treatment of hyperkalemia with cation-exchange resins, such as Kayexalate, has decreased the requirement of dialysis for the sole purpose of treating hyperkalemia. Usual doses are 10 to 50 g by mouth or enema every 2 to 6 hours. Sorbitol as an osmotic cathartic (5 to 10 ml) by mouth or by enema also promotes diarrhea and intestinal potassium losses. Since many drugs are excreted through the kidney, decreased renal function requires decreased doses of most antibiotics and other drugs such as digitalis. Magnesium-containing compounds, such as antacids, should be used sparingly in the oliguric patient because of the possibility of magnesium toxicity.

An oliguric patient should not be kept in the forward area any longer than necessary. Instead, he should be evacuated as expeditiously as possible to a center which possesses an artificial kidney and which is otherwise especially equipped to treat acute renal insufficiency. If he cannot be evacuated, a patient who remains oliguric for 72 hours should be treated by the following emergency measures, designed to reduce or counterbalance an excess of serum potassium:

1. Intravenous glucose is given in 10-percent concentration through the superior vena cava. This measure will cause potassium to be reincorporated into intracellular glycogen and will lower the serum concentration. The concomitant use of insulin may facilitate this process.

2. Since calcium is a specific antagonist of potassium, a continuous infusion of 10-percent calcium gluconate will counterbalance excess potassium if it is not extreme. Although sodium is also an antagonist of potassium, large amounts of this ion

should not be used during the first days of acute renal insufficiency. Sodium, in this setting, is used sparingly and only to replace that lost by urinary excretion, gastric suction, or diarrhea. Generally, one-third normal saline is used to replace urine output, and normal saline to replace gastric fluid loss.

3. Fluid balance is maintained by the use of carefully calculated amounts of the required fluids. Consider, for example, a patient who has excreted 50 ml of urine and who has lost 150 ml of fluid in vomitus or by gastric suction within 24 hours. His measured output is thus 200 ml. This amount should be added to the basic allowance of 500 ml to give a total intake for 24 hours of 700 ml. Of this, 100 ml may be 10-percent calcium gluconate, 400 ml should be 10-percent glucose in water with 10 units of regular insulin, and 200 ml should be isotonic saline. Sodium bicarbonate in 7.5-percent solution may replace saline if the pH determination reveals metabolic acidosis. Since some wounded patients develop respiratory alkalosis, monitoring of serum pH is necessary to determine appropriate fluid replacement. Peritoneal and extracorporeal dialyses are effective techniques of treatment but are not usually feasible outside a special center staffed by personnel trained in these techniques.

Lung

Factors in the production of acute posttraumatic pulmonary insufficiency in the severely injured adult are:

1. Direct trauma to the lung by blast, abdominal injury, explosive decompression, or fractured ribs.

2. Fluid overload, particularly that of intravenous salt solution without colloid.

3. Endotracheal intubation or tracheostomy, which, although sometimes necessary and essential to adequate treatment of the syndrome, may cause undesirable drying of the tracheobronchial tree and may introduce organisms directly.

4. Wound infection with bacteremia and superimposed pneumonia.

5. Severe nonpulmonary trauma.

6. Massive transfusions, for example, 10 or more units of whole blood.

7. High oxygen concentration in the inspired air for prolonged periods.

The typical course of posttraumatic pulmonary insufficiency occurs in several stages. During the phase immediately following wounding, including resuscitation and surgery, embarrassment of respiration may not be noticeable, although some tendency to hyperventilation may occur. This is followed by an interval when the patient superficially appears to be quite well, apart from other injury. This characteristically occurs immediately after resuscitation during a phase of high cardiac output. After a variable interval, measured in days, progressive pulmonary insufficiency follows. This is indicated by progressive anoxemia, often with hypocapnea, refractory to high concentrations of oxygen in the inspired air due to pulmonary venoarterial shunting. Diffuse pulmonary infiltrates, bronchopneumonia, terminal hypercapnea, acidosis, and death may follow.

Fluids must be administered cautiously to patients who have sustained significant direct or indirect injury to the lung. Such patients are notably sensitive to fluid overloads, especially of sodium-containing solutions. Guarding against fluid overload is possible by monitoring central venous pressure. This is most sensitive and accurate if the fluid being administered is blood or plasma. When large amounts of crystalloids are administered, marked overadministration of fluids, to the point of pulmonary edema, can occur even with a normal central venous pressure. Persistent excessively high urine output, that is, over 100 ml per hour for 3 hours or more, is strong evidence of fluid overadministration.

Noxious drying of the lower tracheobronchial tree can be avoided if the patient breathes adequately humidified air. If access to the lower airway is considered essential, endotracheal intubation is used first, reserving tracheostomy for prolonged intubation. In any event, sepsis, drying, and oxygen concentrations over 75 percent should be avoided. Superimposed pulmonary infection, secondary to introduction by direct or hematogenous routes, will aggravate the syndrome and must

be avoided. Therefore, appropriate surgical care of associated wounds is essential.

The treatment of posttraumatic pulmonary insufficiency is less effective than its prevention. With extensive pulmonary shunting, that is, venoarterial admixture from perfusion of unventilated segments of the lung, the oxygen tension in the blood is unresponsive to increased oxygen tensions in the inspired gas mixture. This response, in itself, is a measure of the extent of the shunt. Management of the patient with refractory hypoxemia and flocculent lung densities consists of some or all of the following steps:

1. Mechanical assistance to respiration with occasional use of expiratory resistance to expand the lung. Volume-controlled ventilators are preferable to pressure-controlled devices.
2. Avoidance of oxygen tensions in the inspired gas mixture that are any higher than those absolutely essential to maintain arterial oxygen tensions over 80 torr. Therefore, blood gases must be monitored.
3. Pulmonary physiotherapy and bronchial toilet and suction using strict asepsis. Specimens may be obtained for culture.
4. Selection of the most effective antibiotics, dependent upon culture sensitivities, and use in high doses.
5. Avoidance of excessive medication and of hypocapnea due to overventilation of lung in the hypoxemic patient.
6. Curarization of the occasional patient who requires mechanical control of respiration to ease his respiratory effort. This is particularly useful in treatment of the flail chest.
7. Parenteral albumin and diuretics to reduce pulmonary fluid.

Stomach and Duodenum

The frequent occurrence of stress ulcers in the stomach or duodenum has been noted in the severely traumatized individual, particularly when sepsis is superimposed. The exact etiology of this occurrence is still poorly understood, although it is probably not associated with acid-peptic erosion. That disturbance in the gastric mucous barrier may be involved is suggested. Gastrointestinal hemorrhage of significant degree

is usually the presenting symptom in these patients. The average time of onset of bleeding is approximately 10 days after injury. The site of the ulceration is usually in the stomach and is frequently multiple. Operation may become necessary in these patients when multiple transfusions are required to maintain homeostasis. Both vagotomy and pyloroplasty and gastric resection have been utilized in the surgical treatment. Regional gastric hypothermia with irrigation of iced saline may sometimes be of benefit; when all other measures fail, the use of general hypothermia may be rarely successful. This method consists in lowering the patient's body temperature to 30° or 31° C for 48 to 72 hours. Bleeding usually subsides within a few hours after the temperature has been lowered to these levels when the procedure is successful. Perforation may also complicate a stress ulcer.

Gallbladder

Acalculous cholecystitis may occur in traumatized patients at a time when it is most difficult to diagnose. It presumably develops under the conditions of dehydration, lack of stimulation by oral intake of food, and the effects of drugs, which occur in the patient with trauma which is usually, though not necessarily, abdominal. It may mimic other more common conditions following trauma and may progress to gangrenous cholecystitis with rupture before it is suspected.

Blood

Hypocoagulability may occur in the severely injured individual, particularly when shock has been present. This situation may be caused by one of two mechanisms:

1. Disseminated intravascular clotting with resultant consumptive coagulopathy (consumption of clotting factors from disseminated intravascular microthrombi).
2. Multiple transfusions of ACD-banked blood.

In the latter situation, clotting factors and platelets are deficient in blood more than 3 days old. Excess citrate in

banked blood may aggravate the situation. Laboratory tests reveal prolonged partial thromboplastin time and prothrombin time and a decrease in fibrinogen and platelets. Treatment consists of the use of fresh whole blood or fresh frozen plasma when platelets are not deficient. Heparin has been advised in repeated small doses in treatment of disseminated intravascular clotting, but caution in its use must be exercised in these patients with multiple severe wounds.

CHAPTER XI

Infection

GENERAL CONSIDERATIONS

War wounds are characterized by devitalization of tissues, extravasation of blood, disruption of the local blood supply, introduction of foreign bodies, and contamination with various bacteria. These factors all interact. The devitalized tissue proteins and extravasated blood provide a nutritional pabulum for the support of bacterial growth and thus are conducive to the development of wound infection, particularly the anaerobic type. Edema, a reaction to trauma, soon follows and produces tension beneath the deep fascia with anoxemia from further embarrassment of the local circulation. These events favor the development of infection.

The timelag between wounding and the development of infection, which varies widely according to the circumstances of the injury, represents the incubation period during which bacteria become acclimated to the wound environment and develop the ability to reproduce.

Early, adequate surgery is, therefore, the most important step in the prophylaxis against wound infection. Cleanly incised, living, healthy tissues, with a good blood supply, are best able to combat micro-organisms on their surfaces. Most of the contaminating bacterial species behave as harmless saprophytes and die rather rapidly after removal of dead tissue and foreign debris.

Antibiotic therapy is an important supportive measure employed for the prevention of impending infection or the treat-

ment of established infection, but its role is limited to these objectives. It is not a primary therapeutic measure. Adequate surgery remains the essential step, and the success of antibiotic therapy depends upon its prompt institution and upon its employment according to certain principles or factors.

Infections in wounds of violence remain a continuing and important problem in spite of adequate surgery and prophylactic antibiotic therapy. They continue to pose a serious threat in deep wounds of the extremities, particularly in compound fractures of the tibia and fibula; in wounds in which large masses of muscle are devitalized and the regional blood supply is compromised; and in penetrating wounds of joints and of abdominal, thoracic, and other body cavities which are not properly treated.

Prophylaxis is of the greatest importance. Once infection is established, it may be lethal and it is always costly in terms of delayed wound healing, further destruction of tissue, prolonged morbidity, and delayed restoration of disturbed body physiology.

ETIOLOGIC FACTORS

When infection develops in a wound, one or more of the following factors are usually responsible:

1. Delay in surgical treatment. An increased timelag between injury and definitive surgery may be caused by the tactical situation; by the overloading of existing facilities and personnel so that, after the patient arrives at a hospital, priorities of treatment must be established; or by the presence of severe shock or some other condition which precludes prompt surgery.
2. Inadequate wound excision. This is of primary etiologic importance.
3. Serious vascular injuries resulting in regional tissue ischemia.
4. Inadequate hemostasis at initial wound surgery, with subsequent hematoma formation.
5. Retention of foreign bodies in the wound.
6. Failure to provide adequate drainage.

7. Tight packing of the wound or the use of tight circular bandages or casts.

8. Primary closure of war wounds.

9. Later closure of the wound under tension, over dead spaces, or in a body area in which the blood supply is poor.

10. Inadequate immobilization of the part.

11. Introduction of secondary infection through soaked and stained dressings, fecal contamination, or frequent inspection of wounds.

12. Failure to recognize and treat a perforated hollow viscus or rectal wound.

13. Presence of bacterial contamination which is resistant to antibiotics.

14. Reliance solely on prophylactic antibiotic therapy.

15. Secondary contamination from exposure to personnel who are carriers of pyogenic bacteria.

16. Presence of metabolic disease, such as diabetes, which predisposes to the development and spread of infection.

BACTERIOLOGY

Bacteriologic studies of war wounds have shown that contamination is universal. All open wounds are contaminated at the time of the first examination after injury. Studies have also shown that contamination with more than one species of bacteria is the rule. The types present vary with the geographic location and terrain, the type of debris soiling the wound, the bacteria resident on the skin and clothing at the time of the injury, and the time between wounding and examination.

The various micro-organisms recovered from war wounds fall into three principal classifications, as follows:

1. The anaerobic spore-forming bacilli of fecal origin.
2. The nonsporulating micro-organisms of fecal origin.
3. The micrococci.

For practical purposes, these micro-organisms may be divided into two groups, as follows:

1. Invasive pathogens, which have the power to invade and destroy living tissues. In this group are the toxigenic clostridia,

including *Clostridium perfringens (welchii), C. novyi (oedematiens), C. septicum,* and *C. bifermentans (sordelli)*; the beta-hemolytic streptococci; and the hemolytic, coagulase-positive *Staphylococcus aureus.* The clostridia are introduced at the time of wounding. Hemolytic staphylococci and streptococci also may be introduced at the time of wounding but more commonly are added later, through contact with reservoirs of infection after the casualty reaches the hospital.

2. Local pathogens. In this group are the proteolytic clostridia (chiefly *Clostridium sporogenes, C. bifermentans,* and *C. multifermentans*) and the gram-negative enteric bacilli such as various species and strains of *Aerobacter, Proteus,* and *Pseudomonas aeruginosa (Bacillus pyocyaneus).* The proteolytic clostridia are introduced at the time of wounding. The gram-negative enteric bacilli also may be introduced at the time of wounding but often are introduced at the hospital. Both groups produce liquefaction necrosis of devitalized tissues and blood clot, with exudation.

Clostridium tetani, which is intermediate between the invasive pathogens and local pathogens, usually is introduced at the time of wounding.

Serial bacteriologic studies show that the bacterial flora of open wounds is seldom static but is usually changing or dynamic. One reason for this phenomenon is secondary inoculation of the wound, which increases as the wound ages. Secondary contamination may occur at operation, on the ward, or elsewhere, whenever a wound is examined or handled or is exposed to respiratory droplets, the attendant's hands, dust, or other conditions not completely aseptic. Bacteria most commonly participating in secondary contamination are the beta-hemolytic streptococci and the hemolytic coagulase-positive staphylococci, which are present in the nose and on the hands of carriers. Other participating bacteria which have become increasingly important in wound sepsis in recent years are the gram-negative forms of *Pseudomonas aeruginosa, Aerobacter aerogenes, Proteus vulgaris, Serratia marcescens,* and *Klebsiella* species.

Many of these bacteria produce toxins and enzymes to facilitate their spread through tissue barriers within wounds. Coagulase, fibrinolysin, proteinase, collagenase, hyaluronidase, and

necrotizing enzymes favor the development and spread of wound infections.

Surgical debridement is not germicidal. It represents mechanical, physical cleansing of the wound, and bacteria may remain, therefore, as contaminants. It is significant, however, that the numbers and types of bacteria recovered 4 to 6 days after initial wound surgery are invariably fewer in wounds which have been debrided adequately than in those in which debridement has been inadequate. Particularly important is the fact that, despite the use of intensive parenteral antibiotic therapy, hemolytic streptococci and gram-negative bacilli are frequently present. These findings clearly indicate the limitation of antibiotic agents in effecting a sterile wound.

CLASSIFICATION OF WOUND INFECTION

Before proceeding with the classification of wound infection, certain facts, already stated, should be reemphasized:

1. The bacterial flora of open wounds is dynamic.
2. Analysis of repeated bacteriologic cultures of wounds shows that contamination is universal.
3. Reexaminations indicate that new organisms appear in wounds and old ones disappear.
4. The appearance of new organisms usually is interpreted as indicative of secondary or hospital reinfection, but organisms latent in a wound may appear when a change in treatment is made. The effect of antibiotic therapy may also contribute to alteration in the bacterial flora.
5. Wound infections are still common, in spite of adjuvant antibiotic therapy, but they are not a common cause of death except in burns, peritonitis, gas gangrene, tetanus, retroperitoneal abscesses, and meningitis.

The important consideration with the presence of organisms in a war wound is not what the organisms are but how the wound reacts to their presence. It follows, therefore, that it is necessary to distinguish, by gross inspection of the wound, among contamination, wound suppuration, and invasive infection. The important clinical distinction must be made between the septic breakdown of blood clot and devitalized tissue,

which is a local process, and the invasive infection of living tissue.

By this concept, local wound suppuration is recognized as the consequence of delayed or inadequate debridement of the wound. It may be caused by invasive pathogens, wound pathogens, or a combination of both groups. Suppuration due solely to bacterial decomposition of devitalized tissues is presumably susceptible to termination by adequate cleansing and debridement of the wound.

Invasive infection, on the other hand, implies that bacteria, having established a nidus in a suppurating wound, have invaded healthy tissues and jeopardized the viability of previously sound tissues. Invasive infection, present in association with wound suppuration, may be caused by the hemolytic streptococci, *Staphylococcus aureus,* anaerobic spore-forming bacilli, or a combination of these. Other bacteria, particularly gram-negative bacilli, also may produce invasive infection. In the absence of a pabulum of devitalized tissue or blood clot, hemolytic streptococci are the most likely cause of invasive infection.

Local Wound Suppuration

The appearance of the infected wound varies considerably with the type of infection present. The wound is typically grayish green, and black areas of slough or devitalized tissue may be present. A thin, seropurulent discharge and an odor indicative of putrefaction may also be present. If the nidus of infection is deep, as it is likely to be around a retained foreign body, gas may bubble from the wound surface and a gas abscess may be demonstrable on roentgenograms. The mere presence of gas, however, is not enough for the diagnosis of wound suppuration, since air may have been carried into the wound by the missile.

The wound is not unduly painful or tender. Some swelling of the surrounding tissues is likely. The temperature and pulse are moderately elevated, and the patient looks and feels ill.

Local suppuration is important because, in a small per-

centage of cases, it may develop into spreading gas gangrene. Prompt management is required, including evacuation and drainage of the offending dead tissue, septic hematomas, and fascial plane abscesses. The wound is dressed with fine-mesh gauze and absorptive dressings and is splinted.

Penicillin or one of the broad-spectrum antibiotics, given preoperatively by the intravenous route, will aid in protecting the patient against invasive infection. The initial intravenous dose of penicillin is 1 to 20 million units and of the tetracyclines 0.25 to 0.50 g. Chloramphenicol should be reserved for specific indications as determined by bacterial sensitivity tests. A dose of 2.0 g of tetracycline or more daily for more than 48 hours may result in hepatic complications; a reduced dose is recommended.

Under optimal conditions, granulations begin to appear in 3 to 5 days, and the wound is well on the way to healing. Recurrent suppuration suggests the presence of an undrained nidus of infection, a retained foreign body, or invasive infection.

Invasive Infection

Typical symptoms and signs of invasive infection are both local and general. They include pain and tenderness of the regional lymph nodes, lymphangitis, and systemic signs of infection.

Treatment begins with the intravenous administration of antibiotic agents according to the regimen just described for wound suppuration. It is usually best to defer wide definitive surgical drainage in the presence of an active invasive infection or of cellulitis until 6 hours or more have elapsed since the time the first dose of antibiotic is given. The advantages of antibiotic saturation before surgery are evident in a decreased incidence of chills and fever after operation.

At operation, the wound is opened widely, blood clot is evacuated, and necrotic or gangrenous tissue is excised. Dependent drainage is provided whenever possible. When facilities permit, cultures of the wound, as well as of the blood, should be taken for testing of bacterial sensitivity to antibiotics. Antibiotic therapy is continued after operation.

ANTIBIOTIC THERAPY

Criteria of Administration

Two conditions are essential for antibiotic control of bacterial proliferation in tissues:

1. The antibiotic employed must be capable of destroying or suppressing the etiologic agent or agents concerned; that is, the bacteria present must be sensitive to the antibiotic employed. Usually a bactericidal antibiotic is preferable to a bacteriostatic one.

2. A therapeutically effective concentration of the antibiotic must be in contact with the tissues which harbor the bacteria for a sufficiently long period to allow the cellular defense mechanisms of the body to eliminate the invading microorganisms.

The second of these criteria imposes limitations on the efficacy of systemic antibiotic therapy when the patient is in shock and circulation of the involved area is below normal or entirely inadequate or when the local blood supply is impaired by the presence of a large abscess or of avascular necrotic or fibrotic barriers. In these circumstances, local applications, such as bacitracin (2 percent) or neomycin (1 percent), may be used. Supplemental local antibacterial therapy is also often advantageous in certain body areas in which diffusion of bloodborne antibiotics may be delayed or in inadequate concentration. Conversely, systemic absorption of local antibiotics may cause severe toxic reactions, for example, nephrotoxicity from topical bacitracin or polymyxin B and deafness from neomycin. Sulfamylon also may be useful as a topical antibacterial agent in surface wounds such as burns.

Sensitivity of Wound Pathogens to Antibiotics

Formerly, complex techniques of testing the sensitivity of bacteria to antibiotics hampered the use of these tests, particularly in urgent circumstances. Techniques for testing bacterial sensitivity to antibiotics have been simplified in recent years, so that, even in wartime, it is seldom necessary to employ antibiotic therapy without such preliminary tests.

The following generalizations can be made:

1. Among wound pathogens, the beta-hemolytic streptococcus of human origin (group A) and the clostridial organisms are susceptible to penicillin, the tetracyclines, chloramphenicol, and erythromycin. These bacteria are also susceptible to gentamicin and bacitracin. Anaerobic streptococci are usually relatively susceptible to these antibiotics. Beta-hemolytic streptococci are frequently resistant, and the pathogenic clostridia are consistently resistant to streptomycin, neomycin, and polymyxin.

2. Hemolytic strains of *Staphylococcus aureus,* isolated from wounds at the time of primary surgery, are usually susceptible to penicillin, but subsequent isolations made in hospitals after treatment with penicillin reveal resistance in from 50 to 90 percent of the strains. These resistant strains may or may not be sensitive to the tetracycline group and bacitracin. Staphylococci, for all practical purposes, are resistant to polymyxin and the sulfonamides.

Antibiotic-resistant staphylococci have assumed increasing importance as a cause of postoperative and posttraumatic infections of wounds. Methicillin, oxacillin, gentamicin, and ampicillin are among the newer antibiotic agents useful in controlling resistant staphylococcal infections.

3. Although the gram-negative enteric bacilli are primarily local wound pathogens, they may produce serious infection when they invade the bloodstream, meninges, or urinary tract. They vary in their susceptibility to antibiotics. When an organism is identified only as a gram-negative bacillus, the chance of selecting an appropriate antibiotic is less than 50 percent.

4. *Proteus* species are resistant to most antibiotic agents, including the tetracycline group, streptomycin, penicillin, and polymyxin. Many strains, however, are sensitive to chloramphenicol, ampicillin, cephalothin, or carbenicillin.

Because of the pattern of variable resistance just described, which is both natural and acquired, it is helpful or advisable to test infecting bacteria for their susceptibilities to the various agents. This practice permits the selection of effective agents, though one must bear in mind the limitations of the method of testing by means of commercial sensitivity disks.

When antibiotic sensitivity testing is not available, selection of the antibacterial agent may be made with the help of table 4.

TABLE 4.—*Suggested choices of antimicrobial agents*

Infecting micro-organism	Agent of first choice	Alternative agents
I. Aerobic bacteria		
A. Gram-positive cocci		
1. Staphylococci		
a. Nonpenicillinase-producing	Penicillin	Cephalothin, vancomycin, erythromycin, lincomycin.
b. Penicillinase-producing	Penicillinase-resistant penicillin (e.g., methicillin, oxacillin).	Cephalothin, vancomycin, erythromycin, lincomycin, gentamicin.
2. Streptococci		
a. Pyogenic—groups A, B, C	Penicillin	Erythromycin, cephalothin, ampicillin.
b. Viridans	Penicillin with or without streptomycin.	Ampicillin, vancomycin with or without streptomycin, cephalothin, erythromycin.
c. Enterococci (group D)	Penicillin G with or without streptomycin.	Ampicillin, chloramphenicol, tetracycline.
3. *Pneumococcus (Streptococcus pneumoniae)*	Penicillin	Erythromycin, cephalothin.
B. Gram-negative cocci		
1. *Neisseria catarrhalis*	Penicillin	Tetracycline.
2. *Neisseria gonorrhoeae*	Penicillin	Tetracycline.
C. Gram-negative bacilli		
1. *Escherichia coli*	Ampicillin, cephalothin	Kanamycin, tetracycline, gentamicin, chloramphenicol.
2. *Aerobacter (Enterobacter) aerogenes.*	Kanamycin	Tetracycline with or without streptomycin, gentamicin.

3. *Klebsiella* species	Cephalothin	Kanamycin, polymyxin, chloramphenicol.
4. *Pseudomonas aeruginosa*	Gentamicin	Colistin, polymyxin, carbenicillin.
5. *Proteus*		
a. *P. mirabilis*	Ampicillin	Kanamycin, cephalothin, gentamicin.
b. Other *Proteus*	Kanamycin	Nalidixic acid, cephalothin, carbenicillin, gentamicin.
6. *Serratia* species	Gentamicin	Kanamycin, chloramphenicol.
7. *Alcaligenes faecalis*	Chloramphenicol or tetracycline	Penicillin G.
8. *Salmonella typhi*	Chloramphenicol	Ampicillin, cephalothin.
9. *Haemophilus* species		
a. *H. influenzae*	Ampicillin	Tetracycline, cephalothin.
b. *H. ducreyi*	Tetracycline	Sulfonamides, streptomycin.
10. *Brucella* species	Tetracycline	Chloramphenicol.
11. *Pasteurella* species		
a. *P. tularensis*	Streptomycin	Tetracycline.
b. *P. pestis*	Tetracycline	Streptomycin.
D. Gram-positive bacteria		
1. *Bacillus anthracis*	Penicillin	Erythromycin, tetracycline.
2. *Corynebacterium* species	Erythromycin	Penicillin.
3. Diphtheroid species	Penicillin	Ampicillin, erythromycin.
4. *Mycobacterium tuberculosis*	Isoniazid with or without streptomycin, with or without *para*-aminosalicylic acid or ethambutol.	Pyrazinamide, cycloserine, ethionamide, viomycin, kanamycin, capreomycin, erythromycin.
5. *Listeria monocytogenes*	Erythromycin	Penicillin.

TABLE 4.—*Continued*

Infecting micro-organism	Agent of first choice	Alternative agents
II. Microaerophilic bacteria		
A. Gram-positive cocci		
1. Streptococci		
a. Hemolytic	Penicillin G	Ampicillin, tetracycline, chloramphenicol.
b. Nonhemolytic	Penicillin G	Ampicillin, tetracycline, chloramphenicol.
III. Anaerobic bacteria		
A. Gram-positive cocci		
1. *Streptococcus* species	Penicillin G	Ampicillin, tetracycline, chloramphenicol.
B. Gram-positive bacilli		
1. *Clostridium* species		
a. *C. perfringens*	Penicillin G and tetracycline	Cephalothin, erythromycin.
b. *C. novyi*	Penicillin G	Tetracycline, cephalothin.
c. *C. histolyticum*	Penicillin G	Tetracycline, cephalothin.
d. *C. septicum*	Penicillin G	Tetracycline, cephalothin.
e. *C. sordelli*	Penicillin G	Tetracycline, cephalothin.
f. *C. sporogenes*	Penicillin G	Cephalothin, tetracycline.
g. *C. tetani*	Penicillin G	Cephalothin, tetracycline.
C. *Bacteroides* species	Tetracycline with sulfadiazine	Chloramphenicol, Vibramycin.

IV. Miscellaneous		
1. *Actinomyces bovis*	Penicillin G	Sulfadiazine.
2. *Nocardia* species	Sulfadiazine	Penicillin G.
3. *Fusobacterium fusiforme*	Penicillin	Tetracycline, erythromycin.
4. *Calymmatobacterium granulomatis*	Tetracycline	Streptomycin.

Antibiotic Therapy Regimen

If a wounded man is in shock, the absorption of intramuscularly administered antibiotics is retarded and unsatisfactory. Blood-serum levels after intramuscular injections of procaine penicillin are low, and tissue levels are suboptimal.

The following initial antibiotic regimen is therefore recommended:

1. If the patient is in shock, soluble penicillin G is given intravenously, as soon after wounding as possible, in doses of 1 million to 20 million units. The injection is repeated every 8 to 12 hours, depending upon the multiplicity and severity of the injuries. Tetracycline, 0.25 to 0.50 g, immediately and every 8 hours, should be given concomitantly by the intravenous route. Antibiotics may be given with other intravenous fluids. If allergy to penicillin is a question, cephalothin may be given instead. When circulatory collapse has been controlled, antibiotics may be given by the intramuscular route; when oral intake is permissible, administration of antibiotics by this route is permissible. In soft-tissue wounds in which wound excision has been considered adequate, penicillin alone is recommended.

2. In penetrating wounds of the abdomen, in which risk from threatening peritonitis is high, tetracycline should be given intravenously, 0.5 g at the time of initial surgery, together with penicillin as recommended for patients in shock. This regimen is continued or modified according to the indications.

3. After 3 to 5 days, the patient's condition and the effects of antibiotic therapy should be evaluated, and desirable changes may be made. The selection of antibiotic agents and the period over which they are used frequently must be tempered by the development of allergic reactions, toxic effects, and the emergence of resistant organisms.

4. Supplemental local antibacterial therapy may be used, as has already been pointed out, in certain body areas in which diffusion of a bloodborne antibiotic may be delayed or may be present in inadequate concentration. Most authorities do not use or recommend the intraperitoneal or pleural application of antibiotics except in unusual circumstances. Instead, they rely upon systemic antibiotic therapy.

5. Systemic cephalothin, gentamicin, ampicillin, carbenicillin, chloramphenicol, erythromycin, kanamycin, colistin, and polymyxin usually are reserved for the therapy of established infection. Neomycin or bacitracin is never used parenterally.

Gentamicin is useful in staphylococcal and gram-negative infection refractory to other antibiotics. Chloramphenicol also may be used on strict indication. Because it diffuses readily into the cerebrospinal circulation, it may be the drug of choice in gram-negative bacillary meningitis. The dose is 0.25 to 0.5 g every 6 to 8 hours by intravenous, intramuscular, or oral administration. Methicillin, oxacillin, gentamicin, cephalothin, or ampicillin may be particularly effective agents for treatment of resistant staphylococcal infections. Methicillin is given in doses of 1 g every 4 to 6 hours either intravenously or intramuscularly. Gentamicin is given intramuscularly only at a dosage level of 3 to 5 mg per kg per day in three divided doses, if renal function is normal. Nephrotoxicity and ototoxicity are possible complications. Cephalothin is given intramuscularly or intravenously in doses of 500 mg every 4 to 6 hours. It is used only infrequently by intramuscular injection, however. If bacitracin is used in topical applications for staphylococcal infections of the brain, thorax, and joints, the usual dose is 500 to 1,500 units (0.01 to 0.03 g) per milliliter of diluent.

Neomycin, because of potential severe nephrotoxic effects, is never used parenterally. Polymyxin and neomycin may be employed topically for gram-negative bacillary infection; the dose is 5 mg per milliliter of diluent. Polymyxin, colistin, kanamycin, gentamicin, ampicillin, or carbenicillin may be employed for serious invasive gram-negative infections. Polymyxin and colistin are particularly effective against *Pseudomonas* infections. See table 5 for antibiotic dosage schedules.

CLOSTRIDIAL MYOSITIS

Clostridial myositis (clostridial myonecrosis, gas gangrene), in contrast to simple contamination with clostridial microorganisms, is a true myonecrosis, which is always acute and is usually diffuse. It occurs principally, though not exclusively, in the lower limbs, the buttocks, and the upper limbs. It is

TABLE 5.—*Daily dosage and routes of administration of antimicrobial agents*

Drug	Oral dose	Intramuscular dose	Intravenous dose	Adult or maximum dose per day
Cephalothin (Keflin)		40–80 mg/kg in 4 doses	40–80 mg/kg in 4–6 doses	12.0 g
Chloramphenicol (Chloromycetin succinate)	50–100 mg/kg in 4 doses		50–100 mg/kg in 3–4 doses (10% solution)	*Child:* 3.0 g *Adult:* 4.0 g
Colistin (Coly-Mycin)	6–8 mg/kg in 3 doses (5 mg/ml)	1.5–5.0 mg/kg in 2–4 doses		300 mg
Erythromycins (Erythrocin) (Ilotycin) (Pediamycin)	30–50 mg/kg in 4 doses	10–20 mg/kg in 3–4 doses	40–50 mg/kg in 4 doses	*Oral:* 2.0 g *I.M.:* 600 mg *I.V.:* 2.0–4.0 g
Gentamicin sulfate (Garamycin)		2–3 mg/kg in 3 doses (7 to 10 days)		5 mg/kg
Kanamycin (Kantrex)	50 mg/kg in 4 doses	15 mg/kg in 2–4 doses	15–30 mg/kg in 2–3 doses (2.5 mg/ml)	*Oral:* 4.0–6.0 g *I.M.:* / *I.V.:* } 1.0–1.5 g *Aerosol:* 1.0 g *Peritoneum:* 0.5 g
Lincomycin (Lincocin)	30–50 mg/kg in 3–4 doses	10–20 mg/kg in 2–3 doses	10-20 mg/kg in 2–3 doses	*Oral:* 2.0 g *I.M.:* / *I.V.:* } 1.2–1.8 g
Penicillin G Potassium or sodium	0.5–2.0 million units in 4–6 doses ½–1 hr before meals	20,000–50,000 units/kg in 4–6 doses	20,000–50,000 units/kg in 4–6 doses	20–60 million units (in selected cases, 100 million units)

Semisynthetic penicillins:				
Ampicillin (Amcill) (Omnipen) (Penbritin) (Polycillin) (Principen)	50–200 mg/kg in 4 doses	100–300 mg/kg in 4 doses	100–300 mg/kg in 4–6 doses	*Oral:* 4.0 g *I.M.:* } 8–14 g *I.V.:* }
Methicillin (Dimocillin-RT) (Staphcillin)		100–300 mg/kg in 4 doses	100–300 mg/kg in 4–6 doses	12.0 g
Oxacillin (Prostaphlin) (Resistopen)	50–100 mg/kg in 4 doses	50–100 mg/kg in 4 doses	50–100 mg/kg in 4–6 doses	6.0 g
Polymyxin B (Aerosporin)	10–20 mg/kg in 4 doses	2.5 mg/kg in 4–6 doses	2.5 mg/kg in 2–3 doses (0.4 mg/ml in 5% dextrose in water for endocarditis)	*Oral:* 400 mg *I.M.:* } 200 mg *I.V.:* }
Tetracycline (Achromycin) (Panmycin) (Polycycline) (Steclin) (Sumycin) (Tetracyn) (Tetrex)	20–40 mg/kg in 4 doses	12 mg/kg in 2 doses	10–15 mg/kg in 2 doses (1.0 mg/ml)	*Oral:* } 2.0 g *I.V.:* } *I.M.:* 500 mg

most dangerous in severe wounds and muscle masses, particularly in the massive muscles of the buttocks and thighs. Sometimes only a single muscle is involved, but, as a rule, whole muscle groups are affected, and the infection often spreads rapidly through an entire limb. The bloodstream is seldom invaded until immediately before death.

Although many open wounds of violence are contaminated with clostridia, clostridial myositis is relatively uncommon, occurring in less than 1 percent of all war wounds. It is, however, one of the most serious and rapidly progressive complications of these wounds.

Etiology

Clostridial myositis is most likely to develop in wounds characterized by the following features:

1. Extensive tearing and bruising of the muscles, such as occur with the penetration of high-velocity missiles.
2. Interruption of the main blood supply to the affected part because of major vascular injury or secondary thrombosis.
3. Interference with the blood supply by tight packing of the wound, the prolonged application of a tourniquet, or a tight plaster cast.
4. Compound fractures of long bones.
5. The deep introduction into the wound of unsterile foreign bodies, clothing, or soil.
6. A prolonged timelag caused by difficulties of evacuation, a heavy influx of casualties, or necessary delay in surgery because of the presence of shock.

Pathologic Process and Bacteriology

The end results of the various etiologic factors just listed are anoxia of muscle tissue, with hemorrhage and edema. Breakdown products of necrotic tissue provide the clostridia present with excellent nutriment. The interruption of the blood supply prevents the ready entrance into the anoxic, devitalized tissue of polymorphonuclear leukocytes and other substances important in body defense mechanisms.

Culture of muscle in clostridial myositis usually yields several species of toxigenic clostridia (particularly *Clostridium perfringens, C. novyi,* and *C. septicum*). The common habitat of these species is the soil, but they also are found in the intestines of many animals, including man. Foreign bodies, fragments of clothing, and bits of skin which have been contaminated from these sources provide sufficient clostridia to infect the wounds into which they are driven. Sometimes one of these clostridia is associated with proteolytic forms such as *C. sporogenes* and *C. histolyticum*. Staphylococci, streptococci, and gram-negative enteric bacilli are also likely to be found associated with clostridia in clostridial myositis.

The toxic metabolites elaborated by the anaerobes present, together with other substances produced by their action on muscle, are responsible for the local pathologic changes in muscle and the associated toxemia and anemia.

Clinical Picture and Diagnosis

Gas gangrene is diagnosed almost entirely on clinical grounds. The usual onset occurs 1 to 4 days after injury, though the upper and lower limits may vary from 8 or 10 hours at one extreme to 5 or 6 days at the other. The most striking feature is a rapid deterioration of a casualty who has previously been progressing satisfactorily.

The first warning symptoms and signs are apprehension, circumoral pallor, and rapid pulse. Within a few hours, the patient becomes anxious and frightened, or euphoric, though he remains alert until the infection is far advanced. Listlessness is sometimes apparent. The color becomes grayish and sweating is profuse. With these signs, a rising pulse in a wounded man who has recovered from shock and who gives no evidence of hemorrhage is highly suggestive.

The first definite symptom is the complaint of discomfort, heaviness, or actual pain in the affected limb or the amputation stump. Once established, pain steadily increases in severity and is often the cardinal symptom. Vomiting is a feature in severe cases.

The temperature rises to 100° to 103° F (37.8° to 39.4° C) as the infection advances. In the early stages, the systolic blood

pressure is approximately normal. Later it falls to 60 mm Hg or lower and the diastolic pressure may be too low to be recorded. Anemia and dehydration are common late findings. Icterus is sometimes present also. In inadequately treated cases, toxemia is profound, a hemolytic anemia is the rule, and circulatory failure is common. The signs of toxemia, including vascular collapse, are not corrected by transfusion alone. It is this fulminating course which makes gas gangrene so dangerous and makes its early and accurate diagnosis of such extreme importance.

Pain is the earliest symptom of clostridial myositis and is frequently in excess of that expected by the wound received. The swelling and edema of the affected part are also disproportionate to that expected by the wound received. Examination of the wound may reveal a profuse serous or serosanguineous discharge, sufficient to soak through massive dressings. The discharge may contain gas bubbles, and it occasionally yields large gram-positive rods on microscopic examination.

The subcutaneous fat in the wound is swollen and discolored. The swollen muscle is bulging, dark, and obviously infected, but the extent of involvement, which is the essential criterion of gas gangrene, seldom can be evaluated until operation. The surrounding tissues are swollen and tense.

Although clostridial myositis often is described as emitting a characteristic rotten-meat odor, this is not true. The odor may be sweet and pungent or foul and fetid, depending upon the species of bacteria present. Odor may be lacking entirely when the infection is caused by *C. novyi (oedematiens),* which is a scanty and late gas producer.

The presence of gas is also variable and may not be apparent clinically until the later stages. Gas production is more marked with *C. perfringens* (*welchii*) infection than with other types of clostridia. Gas bubbles may be seen dissecting in the fascial planes on serial roentgenograms of the affected part. When gas forms, it spreads rapidly beyond the confines of the infected areas. In the so-called wet type of clostridial myositis, which is usually a *C. novyi* infection, swelling and exudation are tremendous, but gas formation occurs very late or not at all.

Skin changes are variable and are usually of less extent than the infection in the underlying muscle. In the early stages,

blanching on pressure is typical. As swelling increases, the skin becomes brownish, and marbling from stasis in the subcutaneous veins is evident. Mottled whitish or greenish areas, in which blebs may form, appear. Blackening and sloughing are characteristic with extensive previous damage to the skin.

Muscle changes are observed in three typical phases:

1. The infectious process is characterized by a transition in the color of the muscle from the normal purplish red to brick red, contractility is lost, and the cut surface does not bleed. The fibers are swollen, more prominent than normal, and friable. Gas bubbles may be seen or felt in the muscle.

2. As the disease process progresses, the brick-red color changes to olive green, and the muscle becomes more friable.

3. Finally, the muscle becomes purplish black and glistening and softens to a stringy, pulpy mass.

Differential Diagnosis

The differential diagnosis of clostridial myositis is extremely important because of the radical treatment which it requires and which is not required in the other conditions with which it may be confused.

Clostridial myositis must be differentiated from the following conditions:

1. Anaerobic cellulitis. In this disease, the infection is limited to the subcutaneous tissue and fascia and usually does not involve vital muscle. Gas formation is far greater than in gas gangrene. The brownish and purulent discharge is profuse. Pain and toxemia are not prominent. When the dirty tissue is excised, the muscle underneath, in sharp contrast to the appearance of the muscle in clostridial myositis, appears healthy, contracts normally, and bleeds vigorously.

2. Anaerobic streptococcal myositis. This infrequent disease has a less acute onset than does clostridial myositis. The toxemia is mild until the infection is well advanced. The blood pressure is only slightly depressed and shock is not evident. Locally, a rapidly spreading cutaneous erythema and swelling are observed; the wound is red and malodorous and discharges large quantities of thin, seropurulent fluid. The muscles are

at first boggy and pale, then bright red, and finally dark purple, pulpy, and friable. They contract normally on stimulation and, until late in the disease, they bleed when they are cut. Gas is usually present. Stains of the exudate reveal abundant gram-positive cocci and pus cells.

3. Anoxic gangrene. Anoxic gangrene, which occurs after ligation or failure of repair of a divided major artery of an extremity, is differentiated from clostridial myositis by the history and by the absence of profound toxemia.

4. Other conditions. Secondary hemorrhage or septic thrombophlebitis can be excluded at examination on the operating table. The subcutaneous and deep-tissue emphysema sometimes present in wounds made by certain types of missiles which suck air into the tissues is differentiated by the absence of toxemia.

Prophylaxis

Early, adequate surgery of war wounds remains the primary means of preventing gas gangrene with its threat to life and the mutilating effects of the management required when it becomes established. Wide incision, thorough excision of all devitalized and crushed tissue with deficient blood supply, and removal of all accessible foreign bodies are the principles responsible for the relatively low incidence of this disease in World War II and in Korea and Vietnam. Adequate debridement is particularly important in deep irregular wounds which tend to loculate. All the recesses of these wounds must be drained adequately if clostridial myositis is to be prevented. Such surgery makes possible the elimination of the conditions in which clostridia propagate as well as most of the bacteria present.

Antibiotic therapy is an established adjuvant to surgery in the prophylaxis of clostridial myositis, but it is effective only when combined with adequate surgery. The tetracycline antibiotics and massive doses of aqueous penicillin G possess marked in vitro and in vivo antibacterial activity for the clostridia of gas gangrene. Chloramphenicol, bacitracin, and erythromycin also may be useful. The recommended treatment is the intravenous or intramuscular administration of penicillin G in doses

of 1 million units every 3 hours. The dosage for tetracycline is 0.5 g intravenously every 6 to 8 hours or 0.2 g intramuscularly at the same intervals.

Clinically and experimentally, prophylactic serotherapy against gas gangrene has been found to be without benefit and its use is not recommended. It is again emphasized that gas gangrene is best prevented by early initial wound surgery with thorough debridement and removal of all dead tissue. The surgeon's best prophylaxis against clostridial myositis is allowing the dirty wound to remain open and closing it by delayed primary closure.

Management

Combined treatment by surgical measures and antibiotics must be instituted without loss of time as soon as clostridial myositis is clinically diagnosed. Success depends on early diagnosis and prompt treatment without awaiting laboratory confirmation.

Preoperative preparation is limited to the rapid intravenous infusion of 3 million units of penicillin and 0.5 g of tetracycline, followed by 500 to 1,000 ml of blood. The infusion is kept running during the operation. These measures should not occupy more than 30 to 45 minutes. Further attempts at resuscitation cannot keep pace with the rapid progress of the disease, and they should not be permitted to delay the necessary surgical treatment.

Anesthesia should be planned so that hypotension and hypoxia are avoided. General anesthesia can be induced carefully with small doses of thiopental and maintained with low concentrations of halothane or nitrous oxide and oxygen. If surgery is not prolonged, dissociative anesthesia produced with ketamine is satisfactory. Spinal anesthesia is rarely a method of choice.

Ample exposure of the wound is necessary, and rapid removal of the affected tissue is essential. When the infection is confined to a single fascial compartment, adequate surgical excision of the affected muscle or muscle group may be sufficient. The

excision, however, must be as radical as is necessary. All discolored muscle and any muscle that does not bleed or contract when it is incised must be removed. This may mean removal of an entire muscle from origin to insertion; or complete removal of a whole muscle group; or, if the whole limb is involved in the injury and infection, amputation of the limb. When the infection has extended beyond the practical limits of amputation or disarticulation, the fascial planes and muscle sheaths are freely incised, to relieve tension and promote drainage. If septic shock develops, a central venous line should be inserted and used to monitor venous pressure, cardiac function, and the administration of fluids and electrolytes.

After operation, a slow infusion of blood or of electrolyte is maintained for 24 to 48 hours. Hourly measurement of the urinary output by indwelling catheter is a guide to volume fluid therapy. The output of urine should be kept between 25 and 100 ml per hour. After operation, penicillin is given in doses of 1 million to 3 million units intravenously every 3 hours, and tetracycline is given intravenously in the dose of 0.5 g every 6 to 8 hours or intramuscularly in a dose of 0.2 g every 6 to 8 hours.

The use of serotherapy in the treatment of established gas gangrene is controversial since antibiotics and hyperbaric oxygen therapy have become available. If serotherapy is elected, pentavalent antitoxin should be used intravenously, after a negative skin test; the dose is two to four vials (each vial containing antitoxin in the following amounts: 10,000 units of *C. perfringens;* 10,000 units of *C. septicum;* 1,500 units of *C. novyi;* 1,500 units of *C. bifermentans;* and 3,000 units of *C. histolyticum*). Pentavalent antitoxin also may be given intramuscularly or subcutaneously after hyaluronidase has been administered. If toxemia is not lessened significantly within 4 to 6 hours, as shown by a decrease in the pulse rate, the initial dose of antitoxin may be repeated.

Hyperbaric oxygenation has proved to be an adjunct to proper surgery and antibiotic therapy. The clostridia are strict anaerobes, requiring a low oxidation-reduction potential in their environment to grow and reproduce. When the quantity of the oxygen in the tissue is greatly increased by hyperbaric oxygenation, the oxidation-reduction potential will

also be increased, resulting in an unsuitable environment for the growth of the clostridia. This modality of care should be used to control the further spread and toxemia of the disease preoperatively and as a postoperative adjunct to care. If a hyperbaric operating room is available, the surgery is best performed in this environment. Recommended hyperbaric treatment schedules are as follows:

1. Primary treatment—2½ atmospheres absolute for 90 minutes every 6 hours.
2. Alternative treatment—3 atmospheres absolute for 60 minutes every 4 hours.

Treatment should be continued daily 3 to 5 days after complete resolution of signs and symptoms.

ANAEROBIC CELLULITIS

Anaerobic cellulitis (fasciitis) is a circumscribed clostridial anaerobic infection which affects devitalized connective tissue rather than living muscle. It is characterized by the septic decomposition of tissues already devitalized by trauma; undamaged muscle tissue is not affected. Gas and pus may infiltrate along fascial planes, and necrosis of fascia bathed in pus may occur.

The onset is more gradual than that of clostridial myositis. The period of incubation is longer. Pain is less severe. Toxemia is relatively slight, though the temperature may be septic in type and higher than in clostridial myositis.

When infected and devitalized subcutaneous and fascial tissue has been excised, normal healthy, contractile, freely bleeding muscle is uncovered, and the diagnosis is immediately evident.

The treatment of anaerobic cellulitis is primarily surgical. The four requisites are: early recognition, adequate decompression by long incisions, removal of necrotic and grossly infected tissue, and establishment of adequate drainage. The process is always more extensive than symptoms and external signs suggest, but the distribution of gas in the tissues and the extent of the infectious process are not necessarily closely related. Unless, therefore, other good grounds for the pro-

cedure exist, areas in which gas is detected should not be incised or excised ruthlessly simply because gas is present.

Aqueous penicillin G and tetracycline are useful adjuncts to surgical therapy. The dosages recommended are those used in gas gangrene. Clostridial serotherapy is not recommended. Adequate surgery and antibiotic therapy with hyperbaric oxygenation usually are followed by prompt and complete recovery.

STREPTOCOCCAL MYOSITIS

The management of streptococcal myositis requires a minimum of surgery combined with the antibiotic regimen outlined for gas gangrene. Surgery consists of relaxing incisions through deep fascia and into muscle to provide adequate drainage and relieve tension. Care must be taken to extend the incision beyond the area of obvious infection in the neighboring or adjacent tissues. Radical surgery after incorrect diagnosis in this condition may lead to septicemia and death.

TETANUS

Tetanus, a severe and dreaded infectious complication of wounds, is caused by the action of *Clostridium tetani* and its toxin on nervous tissue. It carries a mortality rate of approximately 50 percent. It is characterized by local and general convulsive spasms of the voluntary muscles. *Clostridium tetani* is a strict anaerobe which exists in spore form in the soil and in the intestine of animals, including man.

Tetanus infection may supervene in an injury in which the damaged tissues have become colonized by tetanus bacilli. Local necrosis, suppuration, or ischemia provides the conditions necessary for the contaminating spores to evolve into their vegetative form and multiply rapidly at the site of infection. Once the vegetative forms have begun to multiply, large amounts of tetanus toxin are produced.

The incubation period (the timelag between wounding and the first evidence of infection) is usually 6 to 12 days, but it may vary from 4 to 21 days or longer. In any event, the incubation

period is sufficiently long to prevent the development of tetanus in war wounds if proper prophylaxis is achieved within a day or two of injury.

The toxin produced by *C. tetani* in a wound diffuses rapidly through the bloodstream or along the nerves in a cranial direction to the major regional nerve trunks, the spinal cord, and the brain stem. Death results from intoxication of the bulbar nuclei.

Clinical Picture and Diagnosis

While it may arise in a wound anywhere in the body, tetanus, like gas gangrene, most often follows injuries of the leg, thigh, buttocks, or axilla. Small, deep puncture wounds, which often appear trivial, are important sources of infection.

Early clinical manifestations may be of a general nature, such as irritability, insomnia, muscular tremors, or local spasm or rigidity in the muscles near the wound. Sore throat, painful dysphagia, stiff neck, and difficulty in beginning micturition may be early evidence of muscular irritability. The dental officer may be the first to see the patient because trismus is mistaken for some oral condition.

Trismus and risus sardonicus resulting from spasm of the masseters and muscles of the face are signs of established tetanus. They are soon followed by arching of the spine (opisthotonos) and respiratory difficulties from laryngeal and intercostal spasm. The contractions are aggravated by spasms whenever any sensory excitation occurs.

The disease proceeds with fever, sweating, and oliguria, while the mind remains clear. Death usually occurs as the result of respiratory arrest during painful, generalized convulsions. Toxemia, pneumonia, hyperpyrexia, and cardiac failure are other causes of death.

The diagnosis of tetanus is a lengthy, difficult procedure, and the morphologic appearance of the organism in stained smears (the so-called tennis-racket terminal spore in a gram-positive bacillus) usually is not sufficient to differentiate *C. tetani* from other terminal-spored anaerobes.

Prophylaxis

The value of thorough debridement of wounds cannot be overemphasized in the prevention of tetanus. In addition, all wounded casualties, whether their injuries are accidental or wounds of violence, should be given passive or active immunization. In wartime, as in peace, tetanus threatens primarily only wounded persons not previously immunized. Experience has shown conclusively that prophylactic active immunization can eliminate tetanus as a complication of wounds. Immunity might be achieved by passive immunization in those previously not actively immunized.

Active immunization.—Active immunization with tetanus toxoid provides the most effective protection. Basic immunity is achieved by two injections of 0.5 to 1.0 ml of alum-precipitated toxoid at 3- or 4-week intervals, followed by a booster dose of 0.5 ml of toxoid 3 or more weeks later. A booster dose also should be given at the time of a subsequent injury.

Once immunity is achieved, the serum of the injured person at the time of infection with spores will neutralize the toxin at once if neutralizing antibodies are present. Otherwise, the stimulus of the toxin to the previously sensitized reticuloendothelial system will evoke sufficient antibodies to protect the patient within a period of time much shorter than the usual incubation time for this disease. The casualty who has been properly immunized, therefore, will be protected adequately against tetanus by the prompt administration of a booster injection of 0.5 to 1.0 ml of toxoid at the time of injury. Toxoid contains no animal serum, and sensitivity reactions are uncommon. Patients with a history of asthma or hay fever are more liable than others to an allergic reaction, but it usually responds to antihistaminic drugs. Recent evidence suggests that a satisfactory antitoxin titer occurs in persons who have received a toxoid booster injection as long as 20 years after their basic immunization or their last booster injection.

Passive immunization.—Passive immunization with tetanus antitoxin provides temporary immunity for 10 to 14 days or a little longer. It is employed, for patients not previously immunized, by a series of two or three tetanus toxoid injections. The routine is as follows:

Human tetanus hyperimmune globulin (Hyper-Tet) is an effective material for passive immunization against tetanus and is recommended instead of equine or bovine antitoxin to avoid the problems of allergic reactions, rapid elimination of the antitoxins, and delayed serum sickness. Human hyperimmune globulin in doses of 250 to 500 units should be given intramuscularly, never intravenously. High circulating antitoxin levels may be obtained with lower doses, and the rate of disappearance is slower than with the use of equine antitoxin.

Combined active and passive immunization.—In patients not previously immunized with toxoid, combined active and passive immunization should be employed after compound fractures, wounds involving joints, lacerations of tendons, and other open dirty wounds. The routine is as follows:

1. The intramuscular injection of 250 to 500 units of hyperimmune globulin (Hyper-Tet).

2. At the same time, an injection of 0.5 to 1.0 ml of alum-precipitated toxoid is given intramuscularly, into a different area of the body, with a different syringe and needle. Generally, the toxoid can be given in one arm and the antitoxin in the other.

3. The dose of toxoid is repeated at 3- or 4-week intervals for a total of three doses. Immunity can be anticipated 2 to 3 weeks after the third injection.

Surgery.—Early, adequate surgical treatment of all wounds is an essential step in the prevention of tetanus. Conversely, conditions readily become suitable for the germination of spores and the production of toxin if debridement has not been adequate and if the wound continues to harbor bits of clothing, blood clot, and areas of necrotic tissue, or if the blood supply is precarious. Tetanus, per se, is not an indication for amputation.

Antibiotics.—Antibiotics should not be relied upon for the prevention of tetanus without the use of active or passive immunization. In the experimental animal, tetracycline and penicillin increase the survival time, but they do not prevent death if they are used alone. They increase the effectiveness of other prophylactic measures against the injection of lethal doses of tetanus bacilli and they are employed, therefore, as in other

conditions, as adjuvants to, but not substitutes for, indicated wound surgery and the use of antitoxin or toxoid.

Management of Established Tetanus

Although the strict application of correct techniques of immunization and surgery has gone far to eliminate tetanus, the infection may still occur in casualties who have not been immunized and in occasional cases in which initial wound surgery has been inadequate. Treatment is aimed at halting the progression of the disease and controlling its manifestations. Tetanus constitutes a true emergency and treatment is always urgent.

The successful management of established tetanus rests largely upon early diagnosis and prompt as well as adequate treatment. Recognition of the disease during the prodromal stage is advantageous. Severe symptoms do not necessarily indicate the fixation of a lethal amount of toxin in the central nervous system. Carefully planned treatment is necessary, and skilled nursing is a very important factor in recovery.

The components of therapy are as follows:

Serotherapy.—Serotherapy is instituted promptly to neutralize any circulating tetanus toxin. No doubt protective antibody levels can be obtained with injection of tetanus antitoxin. Whether or not these protective levels are of benefit to a patient who already has considerable fixation of tetanus toxin in his central nervous system remains debatable. Much of the objection to administering tetanus antitoxin has resulted from the allergic reactions or delayed serum sickness and hepatitis which have followed the use of equine and bovine serum. The current availability of human hyperimmune globulin (Hyper-Tet) for the treatment of tetanus has eliminated many of these objections. Hyper-Tet therapy seems to be as effective as equine or bovine serum, and sustained levels of circulating antitoxin have been obtained with doses of 3,000 to 6,000 units intramuscularly. Prolonged and predictable serum levels for up to 14 weeks after injection have been reported.

Excision of the wound.—Whenever it is practical and anatomically possible, local excision of the wound or incision with

removal of any contained foreign bodies is performed. It is most important that the wound should not be disturbed for at least an hour after the full dose of antitoxin has been given intravenously and has had the opportunity to become effective. This precaution minimizes the risk of absorption of large amounts of toxin released by disturbance of the wound. Local treatment of the widely excised wound may include lavage with hydrogen peroxide or the application of zinc peroxide powder, but neither measure is a substitute for correct surgery or serotherapy.

Sedation.—The patient is placed promptly in a dark, quiet room, and every precaution is taken to prevent unnecessary movements, noise, and excitement.

Drugs helpful in the prevention or control of convulsive seizures include the following:

1. Thiopental sodium. This drug, which is very useful for general sedation, reduces excitability, diminishes muscular spasticity, and decreases the number of convulsive seizures. It is given in a very dilute solution (0.5 to 1 g per 1,000 ml of 5-percent glucose in water) by continuous slow infusion. The dosage and rate of administration should be decreased, or the infusion should be stopped entirely, if deep anesthesia, cyanosis, or evidence of toxicity develops. Should a severe convulsive seizure occur, with respiratory arrest, several milliliters of a 2.5-percent solution of thiopental sodium should be given intravenously immediately. Solutions of thiopental sodium are unstable and should be made fresh daily. No other medication should be added to the solution, and any solution which shows precipitation should be discarded.

2. Other barbiturates. The hypodermic or intramuscular administration of a longer-acting barbiturate, such as phenobarbital or amobarbital (Amytal), may be given at 4-hour intervals alternatively or as a supplementary measure. The dosage depends upon the needs of the individual patient and the severity of the symptoms.

3. Paraldehyde. Paraldehyde may be employed instead of a barbiturate. It is usually given by retention enema, in doses of 10 to 40 ml every 4 hours, as indicated. Occasionally, it can be given by mouth or by indwelling Levin tube in doses of 1 to 4 ml in ice-cold milk.

4. Curarelike drugs. Curarelike drugs may also be used to control convulsive seizures, but experience has shown that they have not prevented death from respiratory arrest.

Tracheostomy.—Tracheostomy may be useful in maintaining a clear airway in patients who undergo frequent episodes of respiratory arrest. In severe cases, the respiratory rate may be controlled with curarelike drugs, a properly monitored volume respirator, and a cuffed tracheostomy tube. A tracheostomy set, endotracheal set, and hand respirator are kept at the bedside.

Nursing care.—Constant nursing care is essential for the patient with generalized tetanus. A special nurse or a physician should be in constant attendance. This is particularly important for the prompt recognition of convulsive seizures with respiratory arrest so that lifesaving treatment may be instituted immediately. The large amounts of saliva and mucus which collect in the mouth and pharynx may be removed by suction through a small catheter passed gently through the nose into the pharynx or through the tracheostomy tube if tracheostomy has been performed. This measure decreases the incidence of atelectasis and pneumonia. A tongue blade covered with gauze inserted between the teeth is kept in place at all times to reduce the possibility of biting the tongue between the clenched teeth.

Intravenous infusions are given using a three-way stopcock to permit the emergency administration of supplemental doses of 2.5-percent thiopental sodium in the event of respiratory arrest. Gavage of a concentrated liquid diet is desirable when this measure can be employed without precipitating convulsive seizures. If deglutition is affected and gavage is not feasible, intravenous alimentation is instituted.

Antibiotic therapy.—Antibiotic therapy apparently exerts no definitely beneficial effect on the course of established tetanus. It is recommended, however, during the course of treatment, to minimize or control the development of pneumonia or secondary invasive wound infections. Aqueous penicillin G, in doses of 500,000 units or more every 12 hours, is given intravenously.

PART III

General Considerations of Wound Management

CHAPTER XII

Sorting of Casualties

Sorting, or triage, implies the evaluation and classification of casualties for purposes of treatment and evacuation. It is based on the principle of accomplishing the greatest good for the greatest number of wounded and injured men in the special circumstances of warfare at a particular time. The decisions which must be made concern the need for resuscitation, the need for emergency surgery, and the futility of surgery because of the intrinsic lethality of the wound. Sorting also involves the establishment of priorities for treatment and evacuation.

GENERAL CONSIDERATIONS

No task in the medical service requires more informed judgment than casualty sorting. The officer responsible for sorting has very heavy responsibilities. He must exercise sound surgical judgment as he decides which patients need immediate resuscitation, which require resuscitation and surgery simultaneously, and which can tolerate delay in surgery. Of equal importance, after the initial surgery, sound surgical judgment is needed in deciding which patients should be evacuated to other hospitals.

The care of the wounded in any battle zone is always influenced by the prevailing tactical situation. These same considerations are reflected along the entire system of evacuation. They eventually affect the type of care rendered in even the generalized or specialized treatment hospital.

Military surgery, however, represents no crude departure from accepted surgical standards. A major responsibility of all military surgeons is to maintain these principles and practices as fully as possible, even under adverse physical conditions. Ideally, all the adjuvants of surgery, including whole blood, plasma, other fluids, chemotherapeutic and antibiotic agents, and anesthetic agents, are available well forward and should be employed in the same judicious manner as in civilian practice. In short, although certain compromises may be necessary, it is indefensible, even in forward areas, not to carry out correct initial measures. If this policy is not observed sedulously, later reparative surgery cannot be performed with the greatest possible benefit to the casualty.

During the Second World War and the Korean war, the first preliminary sorting of battle casualties took place at a battalion aid station. More careful sorting was carried out at the division clearing station, and further sorting at the field and evacuation hospital level. In the Vietnam war, with the advent of rapid evacuation by helicopter from the battlefield, the battalion aid station and division clearing station were frequently bypassed. Casualties were evacuated immediately to a definitive care hospital.

Sorting does not end, of course, with the accomplishment of initial wound surgery at this level. The concept of sorting also includes secondary evacuation to specialty centers and continues through all stages of management until the casualty is either returned to duty or discharged from the fighting forces.

EVACUATION

Under combat conditions, the constant inflow of the wounded exerts a steady pressure upon all medical facilities, while, at the same time, the need to maintain bedspace for additional incoming casualties creates the constant necessity for evacuation of those occupying the beds. To provide ideal surgical management of the individual casualty, close coordination between the professional and administrative services must be maintained.

Medical officers are responsible for the decisions concerning

evacuation of casualties, but they must cooperate realistically with the administrative officers charged with evacuation and hospitalization. These officers, for their part, must adapt movement schedules to the current surgical necessities. Adjustments must be made on the basis of the type and severity of the injuries to be treated, the number of casualties, the work capacity of surgical teams, the existing and possible complications of the wounds, the probable duration of disability, and considerations of delayed recovery and ultimate disability and deformity. Underlying all of these considerations is the basic, immediate, and urgent objective of saving lives.

AIR EVACUATION FROM THE BATTLEFIELD

The availability of rapid transportation by air does not alter in any way the necessity for the correct application of surgical principles. Speed in evacuation and comfort during transportation from the fighting front are highly desirable. Reduction in the timelag from wounding to initial wound surgery will almost always mean the salvage of life and limb, shortening of the period of recovery and rehabilitation, and reduction of functional disability. If, after the initial surgery, accepted surgical principles are not observed in the selection of patients for air transportation, wound infections and deformity and dysfunction will increase without corresponding gain in the effective rates in the Armed Forces.

In Vietnam, the helicopter was used exclusively to evacuate casualties from the forward battle area to definitive treatment facilities. This rapid transportation system markedly increased the survival rate of the seriously wounded. This technique of evacuation should continue to be employed whenever the situation permits. Modifying factors include air superiority, meteorologic conditions, limitations of the casualty-carrying load, and efficiency of ground maintenance services. Helicopter ambulances should be under the exclusive control of medical authorities. The problems of air evacuation are discussed at length in chapter XIII.

SORTING AT THE BATTALION AID STATION

In the Vietnam conflict, the battalion aid station, for the most part, was bypassed with direct evacuation from the battlefield to clearing stations or definitive surgical hospitals. All efforts were expended to resuscitate the critically injured. No patient was immediately considered unsalvageable. After evaluation, only the most severe head wounds were placed in this category. If helicopter evacuation cannot be utilized directly from the battlefield to a definitive surgical hospital, the following criteria will apply:

Group 1. Those whose injuries are so slight that they can be managed by self-help or so-called buddy care. These casualties can be returned promptly to their units for full duty.

Group 2. Those whose wounds require medical care but are so slight that they can be managed at the battalion aid station or in the divisional area. These casualties can be returned to duty after being held for only a brief period.

Group 3. Those whose injuries demand surgical attention (a) immediately, (b) after resuscitation, or (c) as soon as practicable.

Group 4. Those hopelessly wounded or dead on arrival.

As a practical matter, sorting begins with the casualty himself. His wound may be such that he can elect to continue fighting or can walk to the aid station himself, or it may be so serious that he must summon aid.

Sorting is continued by the medical aidman, who must decide whether the wounded man can be directed to walk to the battalion aid station or must be carried to it. He must also decide, before the casualty is evacuated from the frontline, whether immediate treatment is required for relief of asphyxia, control of hemorrhage, or relief of pain. If the casualty is severely wounded, the aidman necessarily begins the first resuscitative efforts.

At the battalion aid station, the casualty is examined by a medical officer or his assistant. Disposition of casualties in groups 1, 2, and 4 may be made quickly. The medical officer should spend most of his time with those in group 3, where treatment will most likely influence the outcome.

SORTING AT THE LEVEL OF INITIAL WOUND SURGERY

At this level, priorities for surgery must be decided. The primary decisions concern the urgency or permissive delay in the provision of supportive therapy and surgical care. These are decisions which cannot be delegated to inexperienced personnel. The officer who makes them must be familiar with the effects of anesthesia and surgery in the special wounds to be treated; with the patient's probable response to resuscitation and operation; with the optimal timing of this operation; and with the immediate postoperative problems.

CONCEPTS OF SORTING

Correct sorting of combat casualties is based upon the following concepts:

1. The mission of the medical service is to support the Armed Forces by providing every facility and skill possible for the care of all casualties.

2. Principles and practices of medical care must be adapted to conditions of combat, whether casualties are seen as individuals or in great numbers.

3. The salvage of life takes precedence over the salvage of limbs, and the preservation of function takes precedence over the correction of anatomic defects.

4. The casualty with a battle wound is in a dynamic, not a static, state. A wound has deleterious effects which continue and may increase until the wound is repaired.

5. As a result of injury, the body responds in such a way as to correct the disturbances caused by the wound. These responses, which are widespread, continue until recovery is complete.

6. The principal mechanical dangers which threaten life after wounding are asphyxia and hemorrhage.

7. Once a casualty reaches a definitive surgical hospital, uncontrolled hemorrhage is the chief immediate cause of death. Although adequate blood replacement usually is accomplished before operation, it may be necessary to operate simultaneously with blood replacement.

PRIORITIES OF TREATMENT

With these considerations in mind, the following priorities for surgical intervention are recommended. Injuries not included in these listings are dealt with according to the indications of the individual case.

1. *First Priority:*

a. Asphyxia, respiratory obstruction from mechanical causes, sucking chest wounds, tension pneumothorax, and maxillofacial wounds in which asphyxia exists or is likely to develop.

b. Shock caused by major external hemorrhage, major internal hemorrhage, visceral injuries or evisceration, cardiopericardial injuries, massive muscle damage, major fractures, multiple wounds, and severe burns over 20 percent. As shock is likely to occur in any of these injuries, it is well to institute treatment to forestall it before it develops.

2. *Second Priority:*

a. Visceral injuries, including perforations of the gastrointestinal tract; wounds of the biliary and pancreatic systems; wounds of the genitourinary tract; and thoracic wounds without asphyxia.

b. Vascular injuries requiring repair. All injuries in which the use of a tourniquet is necessary fall into this group.

c. Closed cerebral injuries with increasing loss of consciousness.

d. Burns under 20 percent of certain locations, for example, face, hands, feet, genitalia, and perineum.

3. *Third Priority:*

a. Brain and spinal injuries in which decompression is required.

b. Soft-tissue wounds in which debridement is necessary but in which muscle damage is less than major.

c. Lesser fractures and dislocations.

d. Injuries of the eye.

e. Maxillofacial injuries without asphyxia.

f. Burns of other locations under 20 percent.

CHAPTER XIII

Aeromedical Evacuation

INTRODUCTION

Aeromedical evacuation is a modern complex transportation system designed to move patients rapidly. Appropriate utilization of this system markedly reduces the timelapse from initial injury to definitive care. That such rapid movement of patients has resulted in overall decreased morbidity and mortality has been demonstrated repeatedly in recent conflicts. This holds true regardless of the category of patients considered.

At the point of initial wounding, medical capability is limited to first aid measures. Aircraft are utilized to provide rapid transfer of the patient to an area providing first-line resuscitation capability. Triage is accomplished at each echelon of medical care. At aeromedical evacuation collecting points, patients are evaluated and categorized as to their need and general stability. From these collecting points, and in awareness of each patient's individual air evacuability, further movements are programed. Patients may be removed at any medical facility en route when it is the professional opinion of the evaluating surgeon that patient safety will be compromised by continued transfer.

AIRCRAFT

Helicopters are versatile, maneuverable aircraft normally utilized to evacuate injured patients short distances rapidly.

The flying time of currently used helicopter ambulances is about 3 hours with a range of 250 to 300 statute miles. Such flights result in the patients' reaching well-equipped medical facilities in minutes. Patients reach operating room facilities with grave injuries that otherwise would be fatal. Such seriously injured patients receive care that would not be possible without the utilization of rapid air transportation.

Other types of aircraft, of the fixed-wing variety, usually bring supplies and personnel into the theater of operations. After offloading, these same aircraft are quickly converted and reconfigured internally to accommodate both litter and ambulatory patients. With the exception of aircraft specifically designed as flying hospitals, most aeromedical evacuation is performed in reconfigured standard military transport aircraft. Aircraft and medical teams are selected carefully in consideration of the patients' needs. Jet-powered aircraft are capable of rapid patient movement in smooth air at high altitude in pressurized comfort. These movements can be accomplished for short or long distances as required. Rest stops are provided along the way, depending upon the distances involved. Such stops are commonly referred to as RON (remain overnight).

SPECIAL CONSIDERATIONS

Individual patients with particular problems require special considerations. Scheduling the evacuation, managing the patient intransit, arranging special attendants and equipment, programing rest stops, and determining appropriate destination hospitals are all vital considerations to the safe, rapid movement of injured people.

The exigency of a given situation may require a patient to be evacuated earlier and for longer distances than ordinarily would be deemed advisable. As a rule, the goal should be to await adequate patient stability before evacuation efforts.

1. *Tracheostomy care:* Tubes should be of proper size. When mechanical respirators are to be used, cuffed tracheostomy tubes may be required. Because of the low humidity of aircraft cabin atmosphere, the use of some humidification device is recommended to avoid the production of dry mucous plugs and to insure proper tracheal care during flight. The ultra-

sonic nebulizer is the most efficient apparatus at this time. A heat aerosol nebulizer is probably the second-best apparatus.

The use of tracheostomy tubes that do not have cleaning cannulae should be avoided. Mucous plugs and encrustations must be removed promptly to avoid respiratory distress and obstruction. Rubber and plastic tracheostomy tubes normally do not have cleaning inner tubes or cannulae. The periodic instillation of 2 ml of sterile isotonic saline solution into the tracheostomy with prompt aspiration enhances the cleansing of the airway.

Endotracheal intubation is usually safer, quicker, more efficient, and well tolerated by the patient. Prompt use of such tubes would eliminate the need for tracheostomy in many instances. Endotracheal tubes should be the first thought, and tracheostomy second, if, indeed, needed at all. An artificial nose attachment, if available, should be placed over the tracheostomy or endotracheal tube during evacuation. This added precaution provides cleaner, moist air to the patient and thus decreases mucous plugging and encrustation.

2. *Cranial tongs:* Special attention should be paid to the proper seating of the tongs. Traction must be maintained by a closed system, preferably with a spring device such as the Collin's spring. In the absence of a spring device, traction may be maintained by heavy rubber tubing tied to the litter frame. To prevent sudden jerking upon the tongs, weights hanging free must not be left attached during flight.

3. *Skin traction:* Stockinette glued to the skin can be utilized to maintain traction during evacuation. The stockinette is incorporated by folding it back (maintaining traction) over and into a plaster cast or by rubber tubing attached to a wire loop incorporated in the plaster. The surgeon who orders the evacuation of the patient is responsible for removing weights and substituting a self-contained traction device before aeromedical transfer. A spring traction device designed for use in evacuation should be used.

4. *Chest tubes:* Preferably, patients should not be evacuated by air with chest tubes in place, nor should they be evacuated within 72 hours after removal of the tube. Absence of pneumothorax must be demonstrated by a chest roentgenogram just before movement. On the other hand, when necessary, chest

tubes may be left in position during evacuation but should be equipped with functioning valves, such as the Heimlich valve. Pressurization of the aircraft to ground level is desirable if such patients must be moved.

5. *Nasogastric tubes:* All patients requiring nasogastric suction at ground level should have such protection during flight. The combination of the basic medical problems, air swallowing due to anxiety and pain, and the reduced barometric pressure at altitude resulting in expanding body gas may cause difficulties. Abdominal pressure under a body cast, pain from distention of hollow viscera, dehiscence, and, most importantly, vomiting and aspiration with serious pulmonary complications may result.

6. *Plaster casts:* The necessity for evacuating patients with circular plaster casts dictates that all such casts should be appropriately monovalved or bivalved before movement. This permits swelling of soft tissue and rapid emergency access to a serious wound beneath the cast. A rapid, useful means for continuing the care of a patient with a plaster cast is attained by appropriate marking on the cast itself. Such inscriptions should include the date and type of injury, the date of surgery and cast application, and a simple sketch of the bone injury. Looking for this vital information scattered in the chart can waste valuable time in an urgent situation.

7. *Vascular injuries:* Patients with vascular injuries require special attention and immobilization. Casts should be bivalved to provide emergency access to the area. When tactical situations permit, primary repair or graft cases should not be transferred for 14 or more days after repair unless the wound has been closed and is healing without evidence of infection. Patients should have the repair date and type of repair inscribed on the cast or dressing.

8. *Stryker frame:* Such frames may be used for transfers by air. Patients should be turned during travel as ordered by the referring surgeon.

9. *Catheter care:* Indwelling catheters in use before transfer should be left in place during transfer. Instructions for specific care en route (both staging areas and aloft) should be provided to the medical teams along the route. Every attempt should be made to maintain urinary output above 1,500 ml per day.

10. *Circulating blood volume:* Oxygenation problems at ground level will be increased at higher altitudes. Patients having hematocrits of 30 percent or below should not be transferred under any but the most urgent situation; if transfer must be accomplished, proper supplies for transfusion should accompany the patient with orders for the use of blood en route. Measurement of pO_2 should be used as a criterion of air evacuability in the seriously ill patient. Levels below 60 mm Hg are considered significant in this regard.

11. *Cerebrospinal leak:* A wound draining cerebrospinal fluid at ground level will drain slightly faster at higher altitudes. These wounds are not a contraindication for transfer if such is desired for other reasons.

12. *Medications:* Certain medications such as antibiotics, narcotics, and analgesics should have a recorded "stop order" to avoid an undesirable extension of this course of therapy. It is essential that the physician ordering evacuation complete the flight tag accurately to assure antibiotic therapy continuation on schedule or discontinuation as required. Some medications are not normally available in standard supply, and when these are to be continued during patient transfer, an adequate supply must accompany the patient.

13. *Burns:* Burn patients may be transferred at any time during their care; however, as in all severely wounded patients, transfer is unwise until the blood volume has been restored and the patient's condition is stable. Early movement, however, following the above recommendation, is preferable to delayed evacuation. Burns greater than 40 percent, or lesser burns associated with severe injuries, ordinarily should have a surgeon in attendance. Preparation for transfer should include:

a. Functioning intravenous pathway.

b. Adequate urinary output.

c. Functioning airway (endotracheal tube or tracheostomy as required).

d. Fresh burn dressings.

e. Complete medical records, particularly accurate fluid-balance sheets.

f. Functioning nasogastric tube.

g. Immobilization of associated injuries as indicated.

14. *Hypothermic blankets:* Do not remove a patient from a

hypothermic blanket and abruptly evacuate him. Equipment is normally available aboard the aircraft to continue such treatment. Convulsions, high fever, and respiratory distress can be expected to develop if this principle is not followed.

15. *Medical attendants:* A medical attendant, if assigned to a particular seriously ill patient, is expected to accompany that patient to the destination hospital.

16. *Dressing changes:* A patient who has had a debridement for a combat wound is considered to have a clean wound. The dressings should not be changed except in an operating room at the time of probable delayed primary closure. Contamination and infection of the wound may occur when the dressing change is conducted under less than optimal conditions. Infection may make delayed primary closure impossible and thus prevent or delay return to duty. Neither odor from, nor staining of, a dressing from blood or serum is an indication for a dressing change. Dressing changes are indicated only for serious complications such as bleeding, unusually high fever, increasing pain, or swelling. The decision for a dressing change should be made by a physician.

CHAPTER XIV

Primary Treatment in the Division Area

Military medical service neither demands nor condones the compromise of professional medical standards. The basic wherewithal to practice medicine of high quality is available to the practitioner on the battlefield. He can provide immediate *onsite* professional treatment to the casualty of war which is usually denied to the victim of a highway traffic accident in civil life. Standard materiel lists for field medical organizations are minimums, not maximums. Every item which is expended in quantity in a busy hospital emergency room can be made available well forward in the division area. Even on long-range patrol, the company aidman can carry crystalloid and colloid intravenous solutions, to be resupplied by his evacuation support. The range of medications which the aidman carries in his bag is limited only by his knowledge of their effective use. Almost invariably, the capability of the company aidman can be increased progressively if his physician supervisor will take the time to teach him, supervise him, and see that he is properly supplied.

CARE AT THE BATTALION AID STATION

The principal function of the division-level medical service is to insure the survival of the patient so that he can be transported safely to a definitive care facility. The availability of rapid medical evacuation by helicopter directly from the battle-

field permits the bypassing of one or more echelons of division medical care. The principles outlined in this chapter must be interpreted in terms of time rather than distance from the site of wounding on the battlefield to the site of definitive care at a forward hospital. Care described herein is appropriate for the battalion aid station, but it may be provided at a division clearing station or even at a forward hospital.

On arrival at the aid station, the casualty should be searched to determine that explosive ordnance has been removed. Sufficient clothing should be removed to allow thorough evaluation and dressing of wounds. Initially, the patient should be insured of an adequate airway, and hemorrhage should be controlled. The time and dose of previous narcotic administration should be determined. The level of consciousness and blood pressure, pulse, and respiratory rates should be recorded and the time noted.

In addition to the general examination of the patient, the functions of medical personnel at the battalion aid station consist of (1) maintenance of cardiorespiratory function, (2) control of hemorrhage, (3) control of shock, (4) application of dressings and splints, (5) control of infection, (6) relief of pain, (7) hydration, (8) recording of all data, and (9) prompt evacuation.

MAINTENANCE OF CARDIORESPIRATORY FUNCTION

Respiratory obstruction or arrest constitutes an emergency of the first order. Air exchange must be maintained while a rapid search is made for the cause of the respiratory difficulty. Speed in beginning resuscitation is vital. If respiration has ceased, mouth-to-mouth resuscitation must be started immediately and continued until respiration resumes spontaneously or can be supported mechanically. A simple mask and bag respirator is the most suitable mechanical device at this level of medical care. The "prone position, back pressure, armlift technique" previously recommended is useful only in situations, e.g. chemical warfare, where mouth-to-mouth resuscitation is not feasible and mechanical respirators are not available. (See Artificial Respiration, p. 167, for techniques of preferred mouth-to-mouth and manual artificial respiratory resuscitation.)

Respiratory Obstruction

When respiratory obstruction is present or seems imminent in an unconscious patient, an efficient airway must be maintained by passing an oropharyngeal or endotracheal tube. In an emergency, any rubber or other flexible tubing of suitable caliber can be used for this purpose. If the tubing is not effective, tracheostomy may be necessary but should be used as a last resort.

Respiratory obstruction may result from a number of causes, in addition to wounds of the chest, as follows:

1. Aspiration of blood, vomitus, or foreign bodies. This is particularly hazardous in an unconscious patient. As much material as possible is removed by suction. Hemorrhage from the nasopharynx should be controlled by packing. A nasogastric tube should be passed if vomiting is present. A tracheostomy may be necessary.

2. Foreign bodies obstructing the entrance to the larynx. These objects are usually broken or loose teeth, pieces of dentures, food, and blood clot. The obstructing foreign bodies should be removed.

3. Edema caused by injury to the pharynx, larynx, or floor of the mouth or caused by inhalation as in burns about the face. An endotracheal tube or tracheostomy may be necessary.

4. Loss of control of the tongue. Obstruction caused by the relaxed and posteriorly displaced tongue is usually easily relieved by a properly fitting oropharyngeal airway. In the absence of such an airway, tilting of the head backwards with a pad under the shoulders, forward traction on the mandible, or placing the patient in the prone position may be effective. A suture passed through the tongue is occasionally necessary and is a simple method of keeping the tongue forward.

Artificial Respiration

Accidents which cause breathing to stop, such as drowning, suffocation, electric shock, head injuries, or poisoning by gases or drugs, can happen at any time; the victim, if unaided, will die from lack of fresh air in his lungs. Artificial respiration,

properly learned and properly performed, can save life in these circumstances. On such occasions, artificial respiration is required without delay. Every second counts. It is wrong to wait for a doctor or other expert. It is not enough to read about artificial respiration. It must be learned and practiced until the right movements become automatic, and then only can one be sure of doing it correctly when the need arises. Without efficiency, it is useless. Artificial respiration should be continued until the victim's normal breathing returns or until expert medical assistance arrives. The preferred and alternative methods are described in the following sections.

Exhaled Air Methods

The oldest and most effective method of artificial respiration, the mouth-to-mouth technique, is superior to all others and is now the method of choice in the U.S. Armed Forces. The principle is the inflation of the lungs with air, much as a bellows inflates a bladder. The rescuer accomplishes this inflation by blowing into the victim's nose or mouth. This method has been shown conclusively to be far superior to any of the manual methods of artificial respiration. It is applicable in all circumstances and at all ages except for casualties with severe facial injuries or those in a contaminated atmosphere while wearing a protective mask. In these rare instances, it may be necessary to resort to a manual method of artificial respiration, and for this reason a description of an alternative manual method has been retained.

Mouth-to-mouth method.—The mouth-to-mouth method (fig. 11) is as follows:

1. Quickly remove heavy equipment and loosen constrictive clothing around waist, chest, and neck.

2. Lay the casualty on his back. Sweep a finger through his mouth and throat to clear away vomitus, foreign bodies, and loose dentures.

3. If available (do not waste time looking for these materials), place a rolled blanket or some other similar material under the shoulders so that the head will drop backward. Tilt his head back so that the neck is stretched and the head is in the chin-up

position (fig. 11A). This alines the air passages so that they do not become blocked by kinking or pressure. If this position can be maintained with one hand, use the other hand to close the nostrils. Otherwise, the operator's cheek is used to close the nostrils.

4. Take a deep breath and open your mouth wide. Seal your mouth around the casualty's mouth and blow forcefully until you see his chest rise. If the chest does not rise, readjust the patient's position to improve airway patency and recheck for other obstruction. Then blow harder, making sure there is no air leakage. The blowing force is appropriately reduced in infants and small children (fig. 11B) to a point at which the chest is noted to expand.

5. After the casualty's chest rises, quickly remove your mouth. Take another deep breath while listening for his exhalation. If his exhalation is noisy, readjust the position of the head and jaw (fig. 11C).

6. When the casualty's exhalation has finished, repeat the cycle. The first 5 to 10 breaths should be deep and given rapidly to provide rapid reoxygenation. Thereafter continue breathing at a rate of 12 to 20 times a minute until the casualty begins to breathe normally. (Caution: Excessively deep and rapid breathing may cause the operator to become faint, to tingle, or to lose consciousness.)

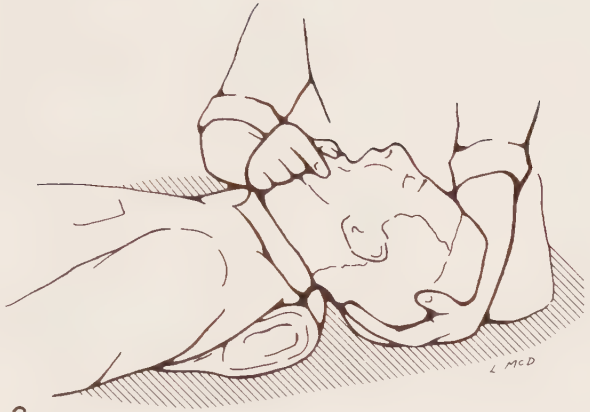
7. If the stomach begins to bulge, overflow air has been blown into the stomach. Apply gentle pressure to the stomach with your hand to attempt deflation. If this maneuver is unsuccessful, the use of a nasogastric tube may become necessary following successful resuscitation.

Mouth-to-nose method.—Spasm of the jaw or injury about the mouth may preclude the mouth-to-mouth method. In this situation, the mouth-to-nose method (fig. 11D) may be used as follows:

1. Remove constrictive clothing and position head as previously described.

2. Tilt the head backwards, place a pad under the shoulders, and grasp the angle of the lower jaw with one hand just below the ear lobe and lift the jaw forcibly forward to pull the tongue forward out of the air passage.

FIGURE 11.—Position of head and neck for exhaled air methods of artificial respiration. A. Proper position of head and neck. B. Make wide seal around mouth and nose and blow gently in children.



C

D

FIGURE 11.—Continued. C. Casualty may exhale better through mouth, so separate his lips to facilitate this. D. Mouth-to-nose method; blow through nose.

3. Seal your other hand over the casualty's mouth to prevent air leakage.

4. Seal your mouth around the casualty's nose and blow forcefully until you see his chest rise.

5. Repeat the cycles as described in the mouth-to-mouth method.

Mouth-to-mouth via artificial mouth airway method.—The mouth-to-mouth method using an artificial airway is similar to the mouth-to-mouth method; one of various types of oropharyngeal airways designed specifically for this purpose may be used. This method may be preferred if an appropriate device is immediately available.

Manual Methods

Effectiveness.—Manual methods are far less effective than the exhaled air methods. Their use should be restricted to those rare cases in which those methods are not applicable; for example, a casualty with severe facial injuries or in a contaminated atmosphere while wearing a protective mask.

Chest pressure-armlift method of Silvester (modified).—The chest pressure-armlift method of Silvester is a manual method of artificial respiration in which the casualty is in a supine position (fig. 12). Its greatest applicability is in a contaminated atmosphere or when facial injuries preclude mouth-to-mouth or mouth-to-nose methods.

The procedure is as follows:

1. Place the casualty on his back with his arms folded on his chest.

2. Clear the mouth of vomitus and foreign bodies by sweeping the fingers behind his teeth and over the back of his tongue. If a protective mask is indicated, it should be applied or replaced as quickly as possible, being certain the mask itself is also cleaned of fluid or vomitus.

3. Aline the positioning of the head and jaw as described on pp. 168–169 under Exhaled Air Methods.

4. Kneel by placing one knee at each side of the casualty's head.

5. Take the casualty's arms just above his wrists and place

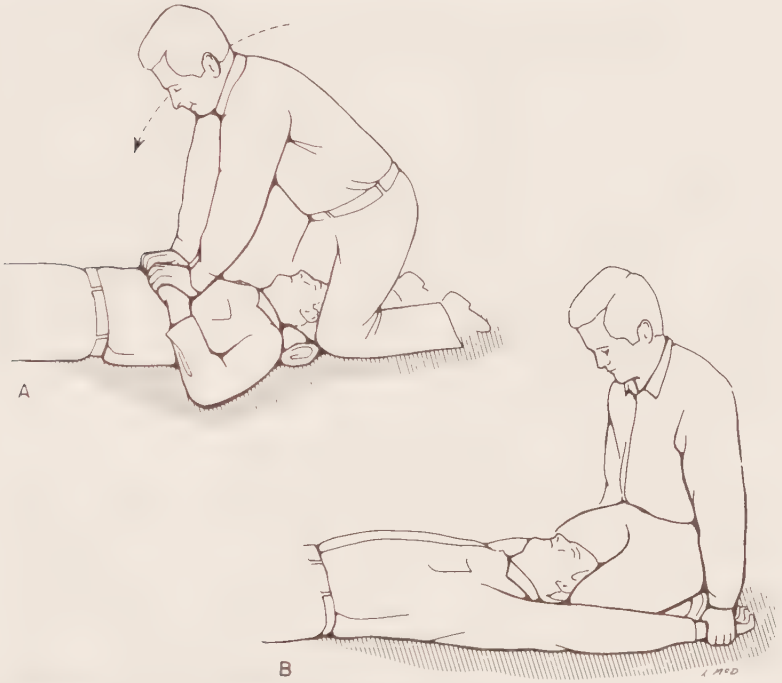


FIGURE 12.—Chest pressure-armlift method of Silvester. A. Expiration effected by steady, uniform pressure on chest. B. Inspiration effected by extending arms backward as far as possible.

them over his lower ribs. Rock forward and exert steady, uniform pressure almost directly downward until you meet firm resistance. This pressure forces air out of the lungs.

6. Move his arms slowly outward from his body and upward above his head. Continue this motion of his arms and sweep them above his head and backward as far as possible. Lifting and stretching of the arms increase the chest size and draw air into the lungs.

7. Slowly replace his arms on his chest and repeat the cycle.

8. The cycle should be repeated 10 to 12 times per minute at a steady, uniform rate to the rhythm of (1) Press—— (2) Lift—— (3) Stretch—— (4) Release——. The release period should be as short as possible.

9. The operator can be relieved by another, the transfer being most expeditiously performed at the "stretch" phase.

External Cardiac Compression

Carrying out artificial respiration is useless if the victim's heart has stopped beating and there is no circulation of blood to carry oxygen. External cardiac compression, as described below, will be carried out only if, after five or six effective inflations of the victim's lungs by the exhaled air method (described in preceding paragraphs), there are:

1. No heart sounds,
2. No improvement in the color of his face and lips,
3. No constriction of his dilated pupils, and
4. No pulse to be felt in his neck or elsewhere.

If one is certain that these conditions exist, external cardiac compression should be started immediately. The technique is simple and easily learned.

With the casualty lying on his back on the ground or on some other firm surface, the operator, with elbows fixed, places the heel of one hand, with the other on top of it, on the lower part of the casualty's sternum (breastbone) in the midline of the chest (fig. 13). Firm pressure is applied vertically downwards, aided by the weight of the operator's body, about 60 times a minute. At the end of each pressure stroke, the hands are lifted slightly to allow full recoil of the casualty's chest. Sufficient pressure should be used to depress the sternum an inch or so towards the vertebral column. It must be realized that the application of too great pressure may cause irreparable damage to internal organs. This applies particularly to children. Artificial respiration must continue simultaneously with external cardiac compression at the rate of about five compressions of the heart to one inflation of the lungs. This can be carried out by one individual, alternately applying the two methods, but is best performed by two individuals working together. Never discontinue artificial respiration in favor of performing external cardiac compression alone. It is far more likely that artificial respiration alone will revive an unconscious patient than external cardiac compression alone. Do not attempt cardiac compression if obvious damage to the victim's chest wall exists.

FIGURE 13.—External cardiac compression.

External cardiac compression should be continued until the patient's heart resumes its normal action and a pulse can be felt at the carotid artery in the neck or the radial artery at the wrist, or until competent medical authority determines that resuscitation is no longer possible. All cases should be evacuated for further treatment.

Chest Wounds

Chest wounds may interfere seriously with respiration. A *sucking chest wound* implies an open pneumothorax. Air entering or being expelled through the wound gives rise to a characteristic sucking sound. Because the chest is open, the

normally negative intrathoracic pressure becomes equal to the atmospheric pressure. The equalization of the intrapleural and atmospheric pressures results in alterations of pulmonary physiology. Therefore, the wound must be sealed immediately by an airtight occlusive dressing. A few layers of petrolatum-impregnated gauze next to the wound, secured by a more bulky dressing, serve well for this purpose. All penetrating wounds of the chest wall must be considered sucking wounds until they are proved otherwise. Often the wound will have been occluded by a dressing before the patient is received at the aid station. If the wound is determined to be satisfactory, it should not be disturbed.

A *tension pneumothorax* exists when the intrapleural pressure exceeds the atmospheric pressure, in contrast to simple pneumothorax, in which the intrapleural pressure is roughly equal to the atmospheric pressure. Tension pneumothorax may occur in penetrating wounds of the chest of the so-called ball-valve type, in which air may enter but not escape from the pleural cavity. However, it is more often seen in lacerations of the lung or bronchus in which the chest wall may be intact. A sucking chest wound may be converted to an "intact chest" by an occlusive dressing, but the risk of the development of the more serious tension pneumothorax should be appreciated.

Tension pneumothorax constitutes a true emergency which requires immediate treatment. Tension pneumothorax can be recognized by the usual signs of pneumothorax, combined with displacement of the trachea and the apex beat of the heart to the opposite side. If a large-bore chest tube with one-way (Heimlich) valve is not available, a large-bore needle (13–15 gage) or any catheter available should be inserted through the second or third intercostal space in the midclavicular line. A flutter valve, made from a finger cot, rubber glove finger, or Penrose drain, should be attached to it.

Flail chest is the result of crush injury to the chest with resulting rib fractures and paradoxical respiration. The unstable chest wall must be splinted. This is best done by firmly strapping the affected area, being sure that the dressing is not circumferential. Sandbags also may be used to support the flail segment. If the flail segment is posterior, merely place the patient supine. The patient must be encouraged to breathe

deeply and cough at this early time, and intravenous narcotics in very small dosage may be beneficial in improving the respiratory effort.

Transportation of all patients with chest injuries should be with the uninjured side up.

CONTROL OF HEMORRHAGE

Most war wounds are complicated by some degree of hemorrhage, internal or external, which, if more than minimal, may impose an immediate hazard to life. Major hemorrhage is usually the result of laceration or severance of a large vessel. It is quite possible for bleeding to be less than alarming after the complete transection of a major vessel because of local vasoconstriction and vessel retraction, whereas bleeding may continue from a partially transected vessel which precludes retraction. While venous bleeding is usually self-limiting, it also may be at times devastating.

Internal hemorrhage can be controlled only by surgery, which is not feasible at an aid station. Until he reaches a hospital, the patient must be kept alive by transfusions of whole blood or by colloid or crystalloid solutions if whole blood is not available.

As an emergency measure, until more effective measures can be instituted, external hemorrhage often can be checked by direct pressure. In most instances, it can be controlled by the application of a firm pressure dressing which must not, however, be so tight that circulation is impeded. Satisfactory control of bleeding by this means may not be immediate, and the medical officer should bear this in mind and should not resort to other less desirable means until pressure obviously has failed.

If pressure dressings do not control the hemorrhage and if the bleeding vessel is visible, a hemostat may be applied to it and incorporated into the dressing. Blind clamping with hemostats is not permissible.

Tourniquets rarely are needed for the control of hemorrhage and should be used only when all other methods fail. A tourniquet properly applied can save life but endanger limb.

An improperly applied tourniquet threatens both life and limb. A common mistake is inadequate compression which fails to occlude the artery but does occlude venous return. This results in an increased rate of blood loss. A tourniquet should be placed as low as possible proximal to the wound. Once in place and adequately controlling hemorrhage, it should not be released until the patient reaches a definitive care facility. The time and site of tourniquet application should be recorded clearly on the field medical card.

CONTROL OF SHOCK

The importance of recognition of established or impending shock and the institution of measures to control it cannot be overemphasized (see ch. IX). Crystalloids, colloids, or blood can be used. Blood typing is not practicable in the division area. O-negative blood usually is available at the division clearing station but not at the battalion aid station. Two or three liters of Ringer's lactate solution given rapidly may temporarily relieve shock and be lifesaving. The amount of intravenous fluids given must be noted clearly to prevent overhydration later in the chain of evacuation.

Large-bore intravenous cannulae should be placed in large veins, preferably in uninjured extremities. The subclavian and external jugular veins are excellent for rapid intravenous administration and can be used satisfactorily for central venous pressure determinations. If a cutdown is required, the saphenous vein at the saphenofemoral junction is a good site. The sterile intravenous connecting tubing is ideal to use as an intravenous catheter because it is larger than most other catheters supplied. With wounds of the abdomen, veins of the upper extremities and neck should be sought first. (Additional details on the management of shock are presented in chapter IX.)

Evacuation should not be delayed longer than necessary, and resuscitative measures must continue during evacuation. Casualties to be evacuated should not be given anything by mouth, regardless of the apparent severity of the wounds. If dehydra-

tion is present, it should be treated only by intravenous fluids, preferably Ringer's lactate.

DRESSINGS AND SPLINTS

Dressings and splints which have been applied by the aidman should be checked carefully for adequacy. If dressings are adequate and if bleeding seems well controlled, they should not be removed at the battalion aid station. A satisfactory dressing should never be removed merely to inspect a wound. Meddlesome redressing is painful, may increase shock or help to initiate it, and introduces contamination.

Fractures are splinted for transport. Many times the upper extremity may be splinted simply by flexing the elbow to 90 degrees in a sling and binding the slung arm snugly but not tightly against the chest. A pad should not be placed in the axilla. The leg may be splinted effectively by a padded wire ladder or by an inflatable splint for short-haul evacuation. The thigh may be splinted, for short hauls, to the opposite intact lower extremity. For longer hauls, when available, traction splints may be utilized. Care, however, must be exercised in preventing pressure necrosis of the dorsum of the foot from the traction straps. The risk of this complication may be diminished by simply leaving the boot in place.

CONTROL OF INFECTION

When a major wound is grossly soiled, or includes major muscle damage, or both, immediate administration of antibiotics is not prophylactic: it is simply early initiation of therapy of a septic process which is already at work (see ch. XI). The administration of a 0.5-ml booster dose of tetanus toxoid intramuscularly in a previously immunized individual prevents the development of tetanus (see p. 146). Administration of tetanus toxoid may be deferred until the patient reaches the next medical installation, but his record should be so annotated. Patients with minor wounds receive toxoid booster in the aid station before being returned to duty.

RELIEF OF PAIN

Contrary to popular belief, a wound is not immediately and profoundly painful. Analgesics should be given sparingly and only to relieve true pain, not to soothe the patient's apprehension or to avert shock. Narcotics are not given to a patient who has significant diminution of consciousness, and they must be used with extreme caution in the presence of any respiratory embarrassment. If narcotics are used, they should be given intravenously to insure absorption, and the time and dose should be recorded clearly on the field medical card.

HYDRATION

Battle casualties frequently are dehydrated when they are received at the aid station. Supplies of water often are limited in the frontlines; and if a casualty is delayed in reaching medical care, dehydration may be extreme, particularly after heavy combat, during the summer, and in the Tropics.

If fluids can be given by mouth, this is the ideal route of administration. However, the presence of an abdominal wound, a severe wound of any sort for which immediate major surgery is required, or shock which impairs gastrointestinal absorption may preclude oral fluids. When in doubt, the patient should receive nothing by mouth. In these cases, intravenous fluids, using Ringer's lactate or normal saline, should be administered.

RECORDING

Ideally, one or more aidmen should have the task of recording information and should be free from patient care duties. The casualty should have a field medical card attached to him which contains his identification and unit; time and mode of injury; diagnosis; condition, including level of consciousness and vital signs; amount, time, and route of all drugs given; time and site of tourniquet placement and other treatment given; and condition on evacuation if resuscitation has been required. These data are important in the later management

of the patient, including timing of surgery. Failure to record them may seriously jeopardize recovery.

EVACUATION

Rapid evacuation to a definitive care facility is a vital factor in reducing combat mortality. Procedures at the aid station must be performed with rapid evacuation in mind.

With availability of helicopter evacuation from the battlefield or the battalion aid station, the decision to fly casualties directly to the nearest supporting hospital depends on four variables: flying time, casualty load, weather, and the tactical situation. In most cases, direct evacuation from battlefield to hospital is preferred. If, however, the flying time to the nearest hospital is significantly longer than to the brigade or division clearing station, casualties requiring prompt resuscitation should receive this at the clearing station before proceeding further. In situations of heavy casualty loads and limited helicopter resources, casualties may be shuttled from battlefield to nearby clearing station from where, after triage and possibly resuscitation, they can be flown to a more distant hospital in order of priority. Even when a hospital is relatively close to the battlefield, the division clearing station may supplement this facility when large casualty loads temporarily inundate its receiving capability. Weather or enemy action may preclude air evacuation.

Litter patients should be transported with the feet forward and the patient supine so that the rear litter bearer can watch for signs of respiratory obstruction. The prone position is rarely indicated, even in the comatose patient. An exception is a patient with maxillofacial injuries when no suction device is available. A semiprone position then might be preferable.

REGIONAL INJURIES

Craniocerebral Injuries

Casualties with head wounds must be observed carefully to determine the degree of neurologic injury and the level of

consciousness. Accurate information obtained at this time, soon after wounding, is essential in the later management of head injuries.

If the patient is conscious, ask whether he is right- or left-handed and record this fact. Warn him not to blow his nose as this would raise intranasal and intracranial pressure and might force infectious material into the meninges through the cribriform plate. Dressings may require changing or reinforcing.

Shock is seldom seen in head injury and, if present, another cause should be suspected.

Morphine should never be given because of its strong central nervous system depressant action. Pain is seldom severe in head injuries. Also, the reaction from morphine may mask valuable diagnostic signs needed to evaluate the patient's condition at the next level of medical care.

If intravenous fluids are started, care should be taken to insure that fluid administration is not excessive. Hydration will aggravate developing cerebral edema.

The patient should be evacuated in a semisupine position with the head turned to one side and the patient turned about 45 degrees. This insures adequate observation and rapid access if respiratory arrest or vomiting occurs.

Injuries of the Spinal Cord

A patient with an injury of the spinal cord should be placed on the litter supine and warned not to attempt to move himself. He should not be moved at all except "all in one piece." This requires several persons, one of whom should control the head. If the cervical spine is fractured, the head should be immobilized. Sandbags placed at the sides of the head serve well for this purpose. Decubitus ulcers have their inception early; therefore, hard objects causing pressure in anesthetic areas should be removed.

Peripheral Nerve Injuries

Peripheral nerve injuries require no treatment in the division area. Unless there is an associated fracture, the extremity need not be splinted if evacuation is prompt.

Maxillofacial Injuries

In maxillofacial injuries, the chief considerations are an adequate airway and the control of hemorrhage. If the injury is severe, tracheostomy may be required. When the wound is dressed, care must be taken to avoid twisting or displacing tissue flaps. Antibiotic therapy should be instituted at the earliest possible time. The patient should be evacuated in either the sitting position or a semiprone position. Transportation in a supine position without adequate means of clearing secretions may prove fatal.

Injuries of the Abdomen

All wounded patients must be examined carefully at the aid station to detect injuries of the abdomen. Particular attention should be given to wounds of adjacent areas. For example, wounds of the chest and buttocks also frequently involve the abdomen.

Gross hemorrhage from the abdominal wall should be controlled, preferably by clamp and ligature. If evisceration has occurred, the extruded organs are covered with sterile petrolatum-impregnated dressings or voluminous moistened sterile gauze dressings. Unless the evisceration is massive, do not attempt to replace the bowel or omentum into the abdominal cavity.

A large intravenous catheter should be inserted into an upper extremity or neck vein; a nasogastric tube should be passed; and an indwelling catheter should be placed in the urinary bladder. Patients with intra-abdominal hemorrhage have a high priority for evacuation. Antibiotics should be instituted, and supportive measures combating shock should be vigorous.

Injuries of the Genitourinary Tract

Most wounds of the lower genitourinary tract involve the external genitalia. For all such wounds, an indwelling urinary catheter should be inserted and hemorrhage should be controlled by either compression or suturing of bleeding areas. All tissue should be preserved. If a catheter cannot be passed and bladder distention is present or develops, a suprapubic cystostomy can be performed simply by inserting a 15-gage percutaneous venous catheter into the bladder, directing the needle through the skin just superior to the pubic symphysis. Supportive measures should be instituted, and evacuation for definitive operation should be prompt.

Injuries of the Extremities

If a vascular injury is suspected, pulses must be documented early. The limb should be wrapped in a bulky dressing and antibiotics begun. The limb should be at the level of the trunk or slightly lower, but never elevated.

For injuries of the hand, treatment at the battalion aid station is limited to the control of hemorrhage and the application of bulky protective dressings. The hand is elevated and bleeding is controlled by steady, evenly distributed compression. All skin flaps must be protected within the voluminous sterile compression dressings. Ideally, these dressings should not be removed until the patient arrives at a definitive care center.

It is important that the hand be dressed and splinted in the position of function. A universal hand splint or a padded, molded wire splint is applied over the dressings on the volar aspect of the forearm and palm, with the wrist joint comfortably dorsiflexed and the fingers flexed over a rolled bandage. A sling is applied, and if the patient is ambulatory, the hand and arm are elevated. If he is recumbent, he is transported with the limb across the chest.

In traumatic amputations, the immediate considerations are control of hemorrhage, prevention or control of shock, and relief of pain. A tourniquet is not used unless it is absolutely necessary. If it is necessary, the tourniquet is applied at the

lowest possible level. The stump should be dressed with a voluminous pressure dressing and the limb should be immobilized in a splint. If the limb is almost severed, completion of the amputation may be necessary after clamping and dividing the remaining pedicle of tissue. The application of a secure pressure dressing over the stump is then possible.

Burns

When a burned patient arrives at the battalion aid station, he is examined rapidly to determine the extent and seriousness of the injuries. All wounds and burns are then dressed with dry sterile dressings. Intravenous fluids should be started and appropriate narcotics given intravenously in small amounts for relief of pain. If the patient shows signs of shock or if evacuation is likely to be delayed, supportive therapy is started according to the standard routine (see ch. III).

TREATMENT IN BRIGADE MEDICAL COMPANY OR DIVISION CLEARING STATION

At the level of the medical installation supporting the brigade—whether it be clearing station, *Hauptverbandplatz*, field ambulance, or *centre de triage*—the management of patients is much the same as it is in the emergency room of a major hospital. Urgent but simple surgical procedures are performed, wounds are dressed, and resuscitation is begun if not previously initiated. When patients are evacuated from the battlefield to a brigade medical station, and thence to a hospital, they should arrive at the supporting hospital in such condition that they may bypass the emergency room and proceed directly to the preoperative ward for consideration of early and timely operation.

CHAPTER XV

Anesthesia and Analgesia

For best results in emergency major surgery for battle wounds, the surgeon must have the assistance of experienced and thoroughly trained anesthetists, either physician anesthesiologists or nurse anesthetists. It is therefore imperative that the best anesthetists available be assigned to the forward surgical units in which lifesaving procedures are accomplished. At this level, professional skill and experience and sound professional judgment are essential.

The most experienced anesthetists, however, may be assisted by anesthetists of more limited training and experience, who can work under supervision. Nurse anesthetists are employed throughout the U.S. Armed Forces and outnumber physician anesthesiologists as they do in civilian hospitals in the United States. Under conditions of stress, other medical officers without special training in anesthesiology can be employed for this purpose if they are properly supervised. Enlisted men should be used only as technical assistants, to take care of equipment, prepare patients, and record vital signs. Throughout the remainder of this chapter, the term "anesthetist" refers to either physician anesthesiologist or nurse anesthetist.

In time of war, anesthetists in advanced surgical units, in addition to administering anesthesia, are called upon to carry out resuscitative measures and to contribute to postoperative care. The success of surgery in acute trauma depends in large part upon the efficiency of these measures. They include evaluation of the patient's physiological status as well as the administration of premedication before anesthesia.

In the wounded who require anesthesia and surgery, the most significant alterations in physiology involve the circulatory and respiratory systems. Since resuscitative measures may have been instituted soon after wounding, the anesthetist, before instituting additional measures, should have a record—for example, field medical card—of the events which occurred from wounding until the patient's arrival at the hospital in which surgical care is to be undertaken. In particular, he must know what fluids have been administered, what other resuscitative measures have been necessary, and what drugs have been given, including the dosage and route of administration of morphine or other narcotics.

During the operation, the duty of the anesthetist, in addition to administering the anesthesia, is to observe and record all physiologic changes which might affect the patient's present condition and influence the postoperative care. He must see that an effective airway is provided and maintained, that bronchial and gastric secretions are evacuated, and that oxygen is administered as necessary. In addition to selecting the anesthetic agents and administering them by the routes selected, he supervises the administration of blood or blood substitutes, electrolytes, and vasopressors during operation; institutes other necessary supportive measures; and makes recommendations for postoperative care.

The anesthetist's responsibility for the direction of these measures continues after operation. He must be certain, before the patient is sent back to the ward, that essential reflexes have returned and that drug depression is at a minimum.

ANESTHESIA EQUIPMENT

The demonstrated capability for more rapid evacuation of seriously wounded casualties from the battle zone has resulted in an increase in the complexity of surgery performed at front-line installations. These changes have necessitated a similar complexity of anesthesia equipment and techniques. Without appropriate equipment, the most experienced anesthetist cannot provide the anesthesia support required for the seriously wounded patient.

Ideally, anesthetic equipment in a forward installation includes standard equipment for the administration of volatile and nonvolatile anesthetics and for intravenous and spinal analgesia, as well as equipment for oxygen therapy and ventilatory support. In addition, an auxiliary air delivery apparatus should be available to be utilized with parenteral drugs such as narcotics, neuroleptics, dissociatives, muscle relaxants, and hypnotics.

Complete endotracheal equipment, including that required for pediatric use, should be available. Appropriate adapters and delivery systems such as circle systems and nonrebreathing systems are essential for anesthetic care. Experience has shown that the anesthetist must be prepared to treat civilian casualties, including substantial numbers of pediatric-age patients.

The modern techniques of medical evacuation have further challenged the skills of anesthesia personnel. Care of the critically injured has required sophisticated monitoring during anesthetic management for the critcally injured soldier. Continuous monitoring of the electrocardiogram, temperature, central venous pressure, heart sounds, and urine output, as well as arterial blood gases and pH, has been essential. The ability to alter the patient's temperature with heating or cooling blankets, as well as the ability to defibrillate, when necessary, has proved invaluable to anesthetic practice.

PREOPERATIVE PREPARATION

The anesthetist, in consultation with the surgeon, must decide when, in the course of resuscitation, it is safe to induce anesthesia and to undertake operation. The judgment as to the efficacy of resuscitative measures and timing of operation in patients seriously traumatized has been detailed previously (see ch. IX).

Aspiration of gastric contents presents a serious hazard in trauma patients. Evacuation of stomach contents by induced or spontaneous vomiting or gastric lavage is advisable before induction of anesthesia. Despite these measures, a cuffed endotracheal tube should be used during anesthesia. Endotracheal intubation may be performed under topical anesthesia before induction.

Preoperative medication consists primarily of atropine, 0.3 to 0.6 mg, usually given intravenously. Morphine or meperidine should be used cautiously in forward areas. They also should be given intravenously because of uncertain absorption by other routes in trauma patients. They should never be used in patients with cranial injuries. Proper doses are 8 to 16 mg of morphine or 50 to 100 mg of meperidine. Barbiturates may be utilized infrequently. They are, however, particularly useful in preparation for regional or local anesthesia. They allay apprehension and minimize hyperactive cortical responses to the anesthetic drug.

TECHNIQUES OF ANESTHESIA INDUCTION

The three categories of anesthesia techniques are local, conduction, and general.

Local Anesthesia

The local anesthesia technique should be reserved for only the most minor of injuries. Lidocaine, 0.5 to 1.0 percent, is the most widely used agent. In situations where a medical facility is acutely overwhelmed with casualties, there is a temptation to do an excess amount of surgery under local anesthesia. Under these circumstances, overdose reactions easily can be encountered. Lidocaine can be administered safely in doses up to 7 mg/kg body weight (see table 6). Good principles of debridement, particularly with high-velocity missile injuries, usually dictate the need for broader anesthesia coverage. Local infiltration seldom suits this need.

Conduction Anesthesia

Conduction anesthesia in combat surgery is an ideal technique. One important consideration is that the busy anesthetist is able to administer safely more than one anesthetic at a time, with monitoring delegated to lesser-trained personnel. Fifteen

TABLE 6.—*Anesthetic agents*

Application, generic and common brand name	Commonly available forms	Recommended maximum dosage[1]
Spinal anesthesia:		
Tetracaine hydrochloride .. (Pontocaine)	1% solution 2 ml ampoule 10 mg/ml 10, 15, 20 mg ampoules powder	15 mg (4 mg for obstetric saddle block)
Lidocaine hydrochloride ... (Xylocaine)	5% solution (2 ml, 50 mg/ml) in 7.5% dextrose	70 mg (40 mg for obstetric saddle block)
Procaine hydrochloride .... (Novocain)	10% solution (100 mg/ml)	150 mg
Nerve block, peridural, infiltration:		
Procaine hydrochloride .... (Novocain)	1% solution (10 mg ml) 2% solution (20 mg/ml)	14 mg kg
Chloroprocaine ..... (Nesacaine)	2% solution (20 mg/ml) 3% solution (30 mg/ml)	14 mg/kg
Lidocaine hydrochloride ... (Xylocaine)	0.5% solution (5 mg/ml) 1% solution (10 mg/ml) 1.5% solution (15 mg/ml) 2% solution (20 mg/ml)	7 mg/kg
Mepivacaine ..... (Carbocaine)	1% solution (10 mg/ml) 2% solution (20 mg/ml)	7 mg/kg
Prilocaine ..... (Citanest)	1% solution (10 mg/ml) 2% solution (20 mg/ml)	8 mg/kg
Topical application:[2]		
Cocaine hydrochloride	4% solution (40 mg/ml)	2.5 mg/kg
Tetracaine hydrochloride .. (Pontocaine)	1% solution (10 mg/ml)	1 mg/kg
Lidocaine hydrochloride ... (Xylocaine)	4% solution (40 mg/ml)	3 mg/kg
Regional intravenous (Bier) block:		
Lidocaine hydrochloride ... (Xylocaine)	0.33% to 0.5% (without epinephrine only)	3 mg/kg

[1] Dosages shown are for use with epinephrine. Reduce dose by 25 to 30 percent if used without epinephrine. For infiltration and nerve-block use, dosage should always be calculated on the basis of milligrams per kilogram of ideal body weight. Spinal dosage should be adjusted after considering body size, physiological status (obesity, pregnancy, age), and requirements of the surgical procedure.

[2] Always used without epinephrine.

or 20 minutes after institution of nerve block or spinal anesthesia, the attention of the anesthetist can be directed intermittently elsewhere without jeopardizing the interest of the patient. Nerve blocks are particularly appropriate for isolated extremity injuries, utilizing lidocaine in 1.0- to 1.5-percent concentration. Spinal or epidural anesthesia is contraindicated in patients in hypovolemic shock but may be administered cautiously when shock has been corrected by appropriate resuscitative measures. Epidural anesthesia offers an additional benefit for the patient with a vascular repair in the lower extremity: chemical sympathectomy, which produces vascular dilatation, can be carried into the postoperative period. Spinal anesthesia is seldom indicated for intra-abdominal exploration. When the integrity of the peritoneum has been violated, the surgeon is obliged to perform wide and detailed exploration. This is uncomfortable to the awake patient unless the spinal anesthetic level is dangerously high. Tetracaine 8 to 12 mg in 0.5-percent concentrate and lidocaine 1.5 to 2.0 percent are the two most widely used agents for spinal and epidural anesthesia, respectively.

General Anesthesia

The requirements for anesthetic agents in traumatized patients are usually far less than under more normal circumstances. This is probably because of two main factors: (1) In low blood-flow situations, the cardiac output is preferentially diverted to the heart, the brain, and, to a lesser extent, the kidney. If the uptake of the anesthetic agent from the lungs remains normal, one can count on achieving much higher levels of anesthetic in these organs at a much faster rate than usual. (2) The decrease in cerebral circulation which might accompany severe blood loss could have some effect on the amount of anesthetic which must be present to achieve a given level of anesthesia. Sudden overdose with either inhaled or injected agents is a potential hazard.

When general anesthesia is the obvious choice for an operative procedure, various agents are usually available to the anesthetist. The final selection usually will depend upon a

variety of existing circumstances. Accordingly, each technique will be considered, highlighting the advantages and disadvantages of each.

Anesthetic Agents

Diethyl ether.—Ether anesthesia has been the mainstay of many battlefield anesthetists, often because it is the only agent available. Despite the multitude of anesthetics currently at hand, ether anesthesia for the battle casualty offers certain indisputable advantages. Trauma, shock, and low blood-flow situations invariably are accompanied by inappropriate distribution of ventilation and blood flow in the lungs. To provide an acceptable level of oxygen in the blood, potent anesthetics which may be delivered with high inspired oxygen concentrations are desirable. Ether is one such agent. In light levels of anesthesia, ether provides more profound analgesia than any other available agent. Thus, the integrity of the peripheral vascular system, particularly the heart, can be maintained while providing satisfactory operative conditions. When relaxation is necessary, ether possesses a unique synergism with curare, calling for doses which are about one-third normal. Such small amounts of curare are unlikely to cause ganglionic blockade which could contribute to lowering of blood pressure. This sensitization of the junctional neuromuscular receptors is equally effective throughout a wide range of ether concentrations in the blood. The slow, stormy induction of anesthesia is invariably absent in the severely injured patient. Various degrees of cerebral hypoxia and decreased reflex irritability usually provide for smooth progress into surgical levels of anesthesia. As previously mentioned, preferential distribution of blood to the vital organs of the body favors early rapid buildup of ether concentrations in the brain. These features which are peculiar to the traumatized patient expedite the induction of anesthesia. When administering light ether analgesia, inspired concentrations seldom exceed 2 percent. Even when combined with oxygen, such concentrations pose no great flammability hazard; however, it would be unsafe to consider any ether anesthetic nonexplosive. In the postopera-

tive period, patients who have received ether anesthesia present fewer care problems. Ventilatory support is seldom, if ever, required and narcotic needs are less. This could be an asset in the busy field hospital.

Halothane.—A sound physiologic basis for selecting halothane in the hypotensive, hypovolemic patient probably exists despite variable experimental reports. Both arterial pressure and cardiac output are somewhat depressed with halothane. The total peripheral resistance is probably unchanged or slightly increased. This seems to occur despite the fact that halothane can relax vascular smooth muscle, reduce central nervous sympathetic vasoconstrictor activity, inhibit ganglionic transmission, and curtail a compensatory increase in catecholamines. Clinically, the dry, pink skin noted in patients under halothane anesthesia is somewhat misleading in that blood flow is preferentially routed to the skin and does not necessarily reflect generalized vasodilatation. Cerebral blood flow, however, also is increased with halothane. In the adequately resuscitated patient, therefore, halothane appears to have a salutary effect in regard to the circulatory system. With deeper levels of anesthesia, however, halothane dilates blood vessels and may improve tissue perfusion in shock. Halothane anesthesia is a definitive and easily reversible method of accomplishing this vasodilatation. Other advantages in the use of halothane anesthesia include the fact that it provides for very rapid induction and emergence, it is nonexplosive, and it is compatible with other drugs found useful in supplementing anesthesia.

Cyclopropane.—Although cyclopropane has many desirable properties in anesthetizing the trauma patient, its high explosive property precludes its use in the combat zone.

Thiopental.—Use of thiopental has few advantages in the care of casualties. It decreases cardiac output by direct myocardial depression, diminishes venous return, and inhibits compensatory reflexes. In light doses, it is antianalgesic, increases undesirable reflex activity, decreases intrathoracic blood volume, and redistributes circulating volume to peripheral areas. It is used to complement other anesthetic agents, particularly to facilitate induction.

Nitrous oxide.—Nitrous oxide is a safe agent when given with adequate oxygen. Under these circumstances, it is not sufficiently potent to be used alone. It is used in conjunction with muscle relaxants, with intravenous anesthetic agents, and occasionally with halothane. This is a preferred technique by many combat anesthetists.

Droperidol.—One of the newer intravenous agents in the field of general anesthetics, droperidol offers some definite advantages in casualty care. Induction of anesthesia with droperidol is rapid and pleasant; the patient quickly becomes detached from his surroundings. The addition of light inhalation analgesics—for example, nitrous oxide—and muscle relaxants provides a complete anesthetic, suitable to any operative procedure. The peripheral vascular effects of droperidol are much like those of chlorpromazine. It decreases peripheral resistance without decreasing cardiac output, thereby enhancing tissue perfusion. Some side effects of droperidol include strong cholinergic stimulation, an effect which is easily counteracted with atropine. In the light of current knowledge, droperidol, plus various amounts of narcotics and muscle relaxants, could be a very reasonable anesthetic choice for trauma and hypovolemia.

Muscle relaxants.—Muscle relaxants frequently are required for various reasons in the care of casualties. The most obvious reason for their use is to supplement an anesthetic which has no relaxant properties of its own or to permit lighter levels of anesthesia with those agents which do provide relaxation. None of the commonly used relaxants is without complications. Succinylcholine recently has been shown to evoke an acute hyperkalemia in patients with extensive trauma, burns, or hemiplegia. Numerous documented cardiac arrests have been attributed to this event. Partial protection is probably provided by the prior administration of a small dose of curare. Patients with shock, burns, and hypovolemia do not metabolize succinylcholine well. Curare, however, is a predictable, effective relaxant. Its pharmacologic effects are altered to some degree by hypothermia and other abnormal body conditions. However, these can be anticipated. The paralysis of curare is reversible, and, as an additional benefit, it is antiarrhythmic in larger doses. One should be careful when administering very

large doses of curare because it is capable of causing ganglionic blockade and hypotension in the hypovolemic patient.

POSTOPERATIVE MANAGEMENT

Consideration is given elsewhere to the many other features of postoperative care. However, in the field of respiratory care, the help of the anesthetist frequently is solicited because of his obvious familiarity with the technical aspects of equipment and his ability to help clinically and chemically in the assessment of respiratory function.

Narcotics should be restricted very carefully after operation and should be used only for true pain. The patient's physiologic balance after operation is often as critical as it was before, and the routine administration of pain-relieving drugs by the clock is both unnecessary and dangerous. Adjustment of pillows, pads, and braces or a change of position will do much to make the patient comfortable. The restless patient may be suffering from hypoxia and not pain.

Immediate problems include the care of the airway, attention to fluid and oxygen therapy, evacuation of secretions, and prevention or control of secondary hemorrhage or shock. It is sometimes necessary to employ bronchoscopic aspiration of secretions causing atelectasis, and to aspirate air and blood from the pleural spaces. Whenever possible, deep-breathing exercises, with changes of position and deliberate coughing, should be used to forestall the development of atelectasis. The respiratory distress syndrome occurring in a small number of trauma cases is described in chapter XXVIII.

CHAPTER XVI

Wounds and Injuries of the Soft Tissues

A primary objective in treatment of battle wounds is localization or isolation of the deleterious effects of injury to permit healing. This objective is best accomplished by the removal of all foreign substances and all devitalized tissue from the wound and the maintenance of an adequate blood supply to the injured part. If these objectives can be achieved, the risk of further local destruction and systemic invasion by pathogenic micro-organisms is reduced to a minimum.

Wound repair is a dynamic process from its inception until all healing processes have been completed. The rapidity of healing is determined in large measure by (1) the timelag between wounding and initial wound surgery, (2) the adequacy of initial surgical care, and (3) the support and protection given locally and systemically to the injured tissues.

PRINCIPLES OF MANAGEMENT

The management of soft-tissue injuries is routinely a two-stage procedure. The first step is thorough debridement of the injured area, after which, with only a few exceptions, the wound is left open. The second step is delayed primary wound closure, preferably within 4 to 10 days after injury. The indication for closure is a clean appearance of the wound.

The first operation is done as soon as the patient reaches an

installation with surgical facilities. Delayed primary closure is usually done in a definitive care facility in the communications zone, but occasionally may be done in a forward hospital when evacuation is delayed. The most important principle in management of battle wounds is their nonclosure following debridement.

Initial wound debridement should be accomplished as early as possible except for delay dictated by patient priorities. When delay is unavoidable, systemic antibiotics should be initiated.

It is taken for granted that all surgery is performed with the patient properly positioned on the operating table as his wounds indicate; that light is adequate; that the surgical field is thoroughly cleansed and correctly draped to permit extension of the operation into areas beyond those immediately obvious; and that the surgical technique will be both aseptic and atraumatic. When possible, a tourniquet should be placed about the limb before surgery of the extremity is undertaken so that it may be tightened immediately. Just to have it near at hand is not sufficient! A working knowledge of the pathways of major nerves and blood vessels is essential for the safe debridement of all wounds.

Antibiotic therapy is only an ancillary measure in the management of combat wounds. Such therapy can never replace adequate surgery performed at the earliest opportunity after wounding. Antiseptics are of doubtful value in the prevention of wound infection. On the contrary, the chemical effect of some may cause further injury to tissues already in jeopardy. However, some observers believe that newer topical antibacterial agents may be of some benefit.

Protective dressings, immobilization and moderate elevation of the limb, supportive measures such as blood and fluid replacement, and all other available supportive therapy are used routinely, with the realization, however, that they are also adjunct measures. Nothing takes the place of sound surgery!

PREOPERATIVE PREPARATION

The patient is evaluated according to principles outlined in chapters VIII, IX, and XIV. A roentgenologic examination is

made in two planes to identify the location of retained foreign bodies.

Antibiotics, to be effective, should be as specific as possible to eradicate the particular sensitive invasive organism. The appropriate antibiotic therapy should be begun as soon as possible after wound contamination. Culture techniques and sensitivity studies are the only means to determine the proper antibiotic for the offending organism. The use of antibiotic treatment in contaminated wounds (in contradistinction to wound infection) is not considered "prophylactic," but rather therapeutic, particularly in war wounds. This means beginning the antibiotic as soon as possible after injury, usually intravenously. If a booster protective injection against tetanus has not been given in the battalion aid station, it is given at this time. Prophylactic sera against clostridial infections have proved to be without value and are not employed.

Among wounds of soft tissues, those with major muscle damage and those for which the application of a tourniquet has been necessary have the highest priority for operation.

TECHNIQUE OF DEBRIDEMENT (WOUND EXCISION)

After the patient has been anesthetized, the clothing is removed or cut away over a large area, and dressings and temporary splints are removed. A sterile gauze pad is held firmly over the wound while the skin over a large adjacent area is thoroughly cleansed, shaved, and prepared for surgery. If the wound is in a limb, the entire circumference is prepared to allow for necessary counterincisions.

Incision

Good exposure (fig. 14A) is required for accurate evaluation of the missile damage and for surgical treatment. Good exposure begins with an incision of skin and fascia sufficiently long to permit adequate inspection of all the wound recesses. Extension of extremity wounds usually should be made in the

longitudinal axis of the extremity. In through-and-through wounds which are relatively superficial, the conjoining bridge of the skin can be cut across and the wound track laid open, the two wounds thus being dealt with as if they were a single wound. If the wound track lies deep, it is better to treat each wound separately. Incision directly over superficial bones, such as the tibia, should be avoided. Incisions crossing joint creases must be S-shaped. As a rule, only a thin border of skin (2 to 3 mm) needs to be excised from the wound edges. Skin is essential for subsequent wound closure, and, fortunately, it has a rich blood supply. If soiled and devitalized skin must be removed, it is best excised en bloc with the subcutaneous fat and fascia, rather than piecemeal.

Steps of Debridement

After the wound has been thoroughly irrigated to remove accumulations of debris, blood clot, and loose foreign matter, it is explored, preferably with the gloved finger, to determine the extent of muscle damage. Renewed hemorrhage from injured blood vessels may occur during this maneuver.

Viable muscle tissue is evident by its color, consistency, blood supply, and contractility. Dead or dying muscle is comparatively dark and mushy; it does not contract when it is pinched nor does it bleed when it is cut. Bold removal of all devitalized muscle is imperative (figs. 14B and C). Deformity or dysfunction which follows extensive removal of devitalized muscle is justified and must be accepted. Failure to remove all devitalized tissue promotes wound sepsis and prolongs morbidity, often making several additional surgical procedures necessary.

The technique of correct debridement can be taught only in the operating room. This is a point which needs to be emphasized in all teaching programs. The following principles are important:

1. During the exploration of the wound with the gloved finger, blood clots, foreign material, and debris free in the wound can be removed. Entrapped accumulations of serum and exudates also can be opened.

2. While complete excision of all devitalized tissue is manda-

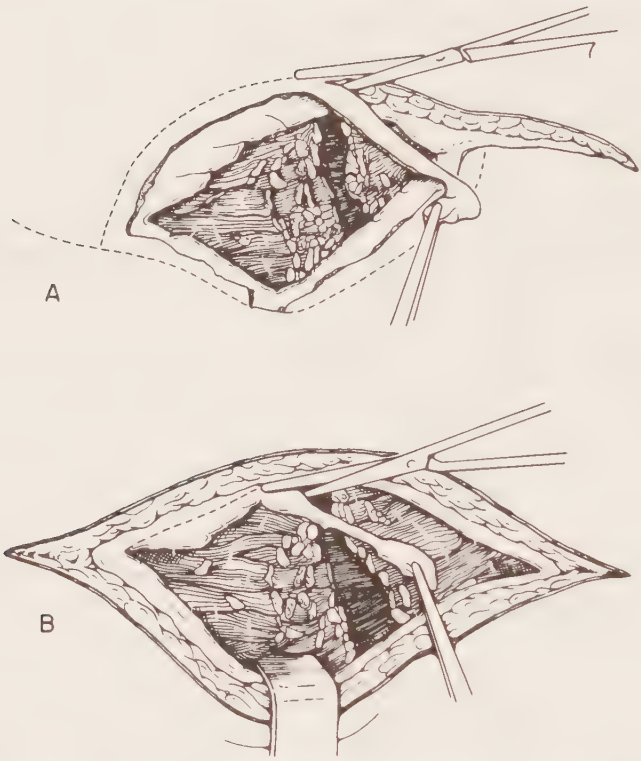


FIGURE 14.—Technique of debridement in soft-tissue wounds. A. Line of incision and excision of traumatized skin. B. Excision of traumatized fascia.

tory, vital structures, such as major nerves and blood vessels, must be protected against damage.

3. All procedures must be carried out gently with precision and skill.

4. Major blood vessels must be repaired promptly.

5. All foreign bodies must be removed, including indriven fragments of clothing; but time should not be wasted seeking elusive metallic objects for which removal would require more extensive dissection. Small detached bone fragments are regarded as foreign bodies and are removed.

6. Tendon usually does not require extensive debridement beyond the grossly destroyed fibers. Loose, frayed edges and ends should be trimmed. Repair of severed tendons should

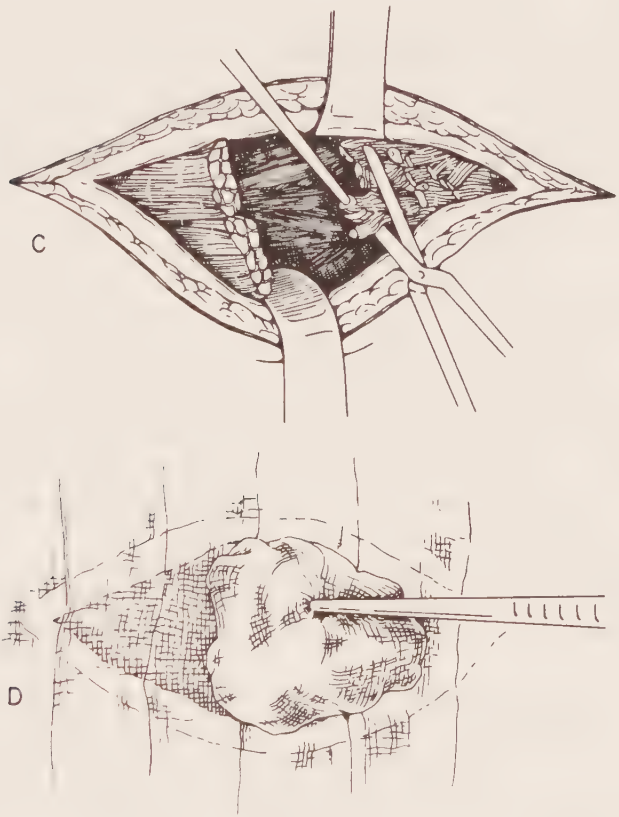


FIGURE 14.—Continued. C. Excision of devitalized muscle. D. Technique of wound dressing.

not be performed during the initial treatment of combat wounds.

7. Hemostasis must be precise.

8. Repeated irrigations of the wound with physiologic salt solution during the operation will keep the field clean and expedite the removal of foreign material and excised devitalized tissue.

9. When the debridement is concluded, blood vessels, nerves, and tendons should be covered, if possible, with soft tissue to prevent maceration and drying.

10. Joint synovium should be closed. If synovial closure is impossible, the joint capsule should be closed (see ch. XVIII). The skin and subcutaneous tissue, in any case, should be left open.

11. Dependent drainage of deep wounds or drainage by dependent counterincision must be employed as necessary.

12. Liberal fasciotomy is often an additional precaution to allow for postoperative swelling.

13. Occlusion packing of the wound is contraindicated, but drainage is facilitated by laying a few wide strips of fine-mesh gauze lightly between the walls of the wound. Fluffed gauze is placed loosely into the pocket thus formed (fig. 14D). Gauze tightly packed into the wound will have the effect of a plug. The dressings on the wound surface should be adequate to protect the wound but should not be constrictive.

14. All wounds must be left widely open, without suture of the skin or deep tissues, with the following exceptions:

a. Wounds of the face (see ch. XXIII).

b. Sucking chest wounds (see ch. XXVII).

c. Head injuries (see ch. XXI).

d. Wounds of joint capsule or synovial membrane (see ch. XVIII).

e. Wounds of peritoneum.

15. Immobilization and correct positioning of the injured part promote healing, and these measures should be employed even if no fracture is present. Immobilization is effected by splints of various kinds, well-padded plaster casts (bivalved), or plaster slabs. Plaster casts which encircle the limb are bivalved as soon as they are applied. This step must never be postponed. The cast is marked with the outline of the wound or fracture and the dates of injury, operation, and encasement.

Dressings on the wound surface should be adequate but not constrictive. Ample absorbent material is laid over the gauze in the wound, and the whole dressing is held in place by adhesive tape applied longitudinally or spirally, bias-cut roller gauze, or elastic bandage. In no circumstances must adhesive strapping be circumferential; it could act as a tourniquet when the limb swells after operation. This precaution is particularly necessary if a plaster encasement has been used.

CRUSH INJURY

Patients who have been crushed beneath debris or run over or whose limbs have been compressed for any reason for an hour or more are likely to develop the so-called crush or compression syndrome. A similar syndrome may occur after widespread damage to muscle even in the absence of prolonged compression. This syndrome is uncommon if not actually rare in peacetime, except in earthquakes, in vehicular disasters, and after cave-ins.

Pathogenesis

The following facts concerning the crush syndrome should be considered:

1. A patient in shock with crushed tissues may be suffering as well from other severe injuries which are likely to cause shock.

2. After release from pressure, the affected part swells because of the extravasation of plasma and blood cells from damaged capillaries. When this happens, oligemic shock is precipitated or aggravated.

3. Oligemic shock which remains untreated will result in hypotension and may go on to acute renal insufficiency (see ch. X).

4. Intracellular substances from damaged muscle, when it is released from compression, leak into the general circulation. Muscle pigment, potassium, creatinine, and lactic acid are liberated.

The part played by pigment deposition in the kidneys remains to be clarified. It once was believed that pigment deposition, per se, caused renal damage and anuria. It now is believed that other factors are also operative.

Clinical Considerations

A clear history of crush injury is not always obtained in wartime, and the syndrome sometimes develops insidiously in patients who appear well when they are first seen. Crush

injuries of the trunk and buttocks will be overlooked if a complete physical examination is not performed.

Although the compressed region may appear normal when it is released from pressure, paralysis caused by the compression is sometimes present. Erythema may appear early after release, at the margin of the affected area, and the adjacent skin may blister. These signs are sometimes the first evidence of damage.

Shortly after release from compression, swelling caused by extravasation of plasma appears in the part. The loss of plasma initiates or aggravates oligemic shock, and the patient's condition rapidly deteriorates. The blood pressure, which was at first maintained by vasoconstriction, falls rapidly as plasma loss continues. The damaged part, which is usually a limb, becomes swollen, tense, and hard; if it is incised, serous fluid oozes from it. The distal pulses tend to disappear. When the fascia is opened, swollen or friable muscle, which in the later stages is pallid, bulges out. Later symptoms and signs may include anorexia, hiccups, dryness of the tongue, and drowsiness or mental disturbances as the blood urea and blood pressure mount.

In favorable cases, diuresis ensues 6 to 8 days after injury and the patient improves clinically, though renal dysfunction may continue for months. In less favorable cases, death may occur promptly from shock or from fluid retention, which is often caused by the unwise forcing of fluid in the presence of renal failure and pulmonary edema. Later deaths are attributable to cardiac arrest caused by hyperkalemia or uremia. In untreated cases, renal insufficiency almost always occurs within a few hours of release of compression.

The first urinalysis, because the urine was excreted before injury, may be normal. Later, the urine becomes scanty and strongly acid. It contains albumin, benzidine-positive pigment, and hollow pigmented casts. When the syndrome is untreated, the urine becomes scanty, dilute, and almost neutral. It also contains albumin and casts. Progressive retention of nitrogen, potassium, and magnesium in the blood occurs. Rapidly occurring hemoconcentration is verified by hematocrit readings.

Local Management

The early splinting of major soft-tissue injuries and fractures is urgently important in crush injuries to minimize hypotension. To reduce tissue metabolism, the limb is kept cool by exposure to air. Unnecessary dressings and unnecessary movements of the limb are hazardous; they cause the release of deleterious substances, particularly potassium in potentially lethal amounts, into the bloodstream.

A tense and swollen limb should be decompressed immediately by liberal incision of the fascia. This measure is particularly urgent when the pressure of extravasated fluid impairs circulation. The application of pressure bandages after release of the compression has been advocated, but experience does not indicate that this is an effective means of preventing the crush syndrome. Amputation of a limb which has been subject to compression is not indicated because of the effects of compression alone.

General Management

The patient who has suffered a crushing injury often has other severe injuries in addition to tissue damage from compression. He will die from shock unless lost plasma and blood are replaced promptly. The objective is to restore the circulatory volume before the blood pressure becomes depressed. Central venous pressure determinations are helpful in prevention of overhydration and in monitoring volume replacement (see ch. IX). Pain and anxiety should be relieved by the administration of suitable drugs.

An indwelling catheter is inserted whenever renal damage is suspected. If the excretion of urine, as measured by the catheter, is as little as 25 ml per hour, the patient, if he is able to swallow, is given alkaline fluids by mouth; otherwise, he is treated parenterally. Alkalinized mannitol solutions or other diuretics such as furosemide are used to maintain a urinary output even as attempts to maintain plasma volume continue.

The blood pressure is determined frequently. Electrocardio-

graphic studies, if available, not only are important prognostically but are also guides to ideal therapy. If possible, the patient should be weighed daily; a progressive increase in weight means that fluid retention is occurring.

The therapy outlined is useful only if it is initiated promptly. If renal failure develops, sharp reduction in fluid administration is indicated, and frequently renal dialysis may be required (see ch. X).

The clinical features of crush syndrome may become evident over a period of days, just as the patient is ready for evacuation on the basis of his other injuries. If renal insufficiency seems to be developing, he should be evacuated, as soon as the other injuries permit, to a medical installation which is capable of investigating and treating the condition with renal dialysis.

MANAGEMENT OF INFECTED WOUNDS

Infected wounds are seldom seen in forward hospitals in which initial wound surgery is performed. When they are encountered, the extensive debridement just described is replaced by a more limited procedure. The aim is simply to relieve tension, insure drainage, and evacuate infected hematomas. This is accomplished by laying the wound widely open and removing all necrotic tissue and all accessible foreign bodies. Vigorous antibiotic therapy is instituted, and parenteral fluids are administered according to the indications. Contamination of war wounds is inevitable. Unless special precautions are taken, subsequent infection will occur in wounds of the face which communicate with the oral cavity and in wounds of the buttocks and perineum, in which the vicinity of the anus and rectum precludes keeping the wounds sterile. Massively infected wounds should be controlled before evacuation.

POSTOPERATIVE MANAGEMENT AND EVACUATION

The limb should be kept moderately elevated after operation and throughout evacuation. Fluid and blood replacement and

antibiotic therapy are continued according to the indications. When the patient's general condition permits, which is usually no sooner than 24 hours after operation, he is evacuated to the communications zone. Delayed primary wound closure, which is the second stage of the two-stage initial wound surgery, is performed there at the appropriate time. As noted previously, patients with crush injury may require earlier evacuation for continuing treatment of acute renal failure.

CHAPTER XVII

Vascular Injuries

Advances in vascular surgery have resulted in a significantly reduced rate of amputation of limbs and in the salvage of life following trauma. The proportion of battle casualties sustaining vascular injuries is relatively small and has been reported to be between 1 and 2 percent in recent wars. Dramatic results following vascular repair by well-trained surgeons can now be anticipated. However, reconstructive vascular surgery may be performed only when proper surgical facilities and personnel are available. To perform such operations at times when surgical facilities are strained, when large numbers of casualties with serious wounds, including vascular wounds, are being cared for, is to prejudice the results in these patients. In addition, exquisite judgment is necessary in evaluating a limb which demonstrates injury great enough to warrant immediate amputation rather than ligation or repair.

Injuries to major vessels usually require prompt surgical intervention if the tissues supplied by these vessels are to be salvaged. The diagnosis should be recognized and preliminary management begun in the field. These patients carry a high priority for evacuation and operation at a definitive surgical center. The majority of acute vascular injuries requiring surgical repair involve peripheral vessels. Few patients with injuries of major vessels of the abdominal or thoracic cavities survive to reach a hospital. Recent advances in rapid evacuation by helicopter, however, have allowed some of the most severely wounded patients to reach the hospital alive. Similar patients in past wars have expired awaiting evacuation.

GENERAL PRINCIPLES

Military experience can differ greatly from civilian experience. An understanding of the high-energy cavitational effects produced by high-velocity missiles upon tissues is important to any surgeon who treats patients so injured. This temporary cavitational effect can cause thrombosis of an artery even though the missile does not actually strike the artery. The mechanism consists of disruption of the intima, arterial wall dissection, intimal prolapse, obstruction of the lumen, and thrombosis. In the combat zone, the number of wounds from high-velocity agents is higher than that in civilian experience.

Although vascular surgery is usually urgent, it must not be done precipitously. It is equally important that every surgeon confronted with the repair of a vascular injury consider the possibility of other wounds and the basic principles of their management.

The patient's general condition must be surveyed rapidly and evaluated properly before reparative vascular surgery is attempted. The majority of the patients with injuries of the major vessels are in poor condition and frequently have sustained multiple injuries. Priority of care of each injury must be ascertained. In addition, the capacity of the patient to tolerate the additional operative time required for vascular repair must be assessed carefully. Failure to observe these basic precautions may result in the loss of a life while the attempt is made to save a limb.

DIAGNOSIS

Injuries to the blood vessels consist of several types: lacerations; transections; avulsions; contusions of the vessel wall with or without intimal laceration, resulting in spasm, thrombosis, or embolic occlusions; expanding hematomas of the vessel wall; and extravascular compression and/or displacement from a hematoma or a bony fragment.

The differential diagnosis of vascular injury is sometimes difficult. A cold, pulseless limb may result from arterial injury or from exposure, shock, spasm, crush injury, or contusing

blows to the extremity. An accurate diagnosis may not be possible until exploration is undertaken. A wound in the vicinity of a large vessel always should arouse suspicion of vascular injury. The following signs and symptoms may be taken as presumptive evidence of arterial damage:

1. The affected extremity may be pale, waxy, mottled, cyanotic, and cold.
2. Pulse may be absent. However, the presence of a distal pulse does not rule out an arterial injury.
3. Analgesia, loss of voluntary motion of the extremity, muscle spasm, or contracture may be present.
4. External hemorrhage, such as obvious spurting of bright red blood, may or may not be present.
5. The affected limb may be larger than the intact limb, particularly in the presence of a large subfascial hematoma.

Extreme caution should be used in diagnosing arterial spasm without exploration of the involved artery.

SURGICAL TIMING

Regardless of the type of injury, the objective of management is restoration of the arterial flow at the earliest possible moment. When the timelag from injury to repair is increased, the failure rate of arterial repair is higher. The best results are obtained within 6 to 10 hours of injury. Since it is impossible to name the precise hour beyond which successful repair can be expected, no inflexible limit should be set beyond which arterial repair is not attempted.

Some patients with injuries of the peripheral arterial system may develop pulsating hematomas or arteriovenous fistulas as a chronic complication of the acute injury. If such a complication is manifest in the early hours after injury, limb viability depends upon the available collateral circulation. Whether or not operation can be delayed safely is wholly dependent upon this factor. Irreversible muscle changes may occur within a few hours if collateral circulation also has been destroyed or is otherwise deficient. Therefore, many variables make impossible the determination of a precise time limit beyond which repair is futile. Since it is not always possible to determine the

irreversibility of ischemic changes in a limb by clinical means, repair of injuries to major blood vessels usually should be attempted when reversibility of the damage is questionable. Even though the limb may not be salvaged, the line of demarcation may be lowered or the joint may be saved.

TREATMENT

Control of Hemorrhage

As a rule, the prolonged use of a tourniquet results in amputation. If a tourniquet is in place, an attempt under proper precautions should be made to replace it with a pressure dressing at the earliest opportunity.

Direct pressure over the traumatized artery usually provides adequate temporary control of the hemorrhage. An anatomic approach to provide adequate exposure to the injured vessel should be employed regardless of the location of the wound. In large wounds, however, the ends of the artery may be visible or readily accessible and can be controlled directly, although a basic principle in vascular surgery dictates first proximal, then distal, control of the artery. Temporary occlusion of the artery can be maintained with tapes or clamps. While the exposed artery is kept moistened with saline solution, dissection should be completed toward the site of injury.

Noncrushing vascular clamps, if available, should be used. However, if crushing clamps are utilized to obtain temporary control of the hemorrhage, they should be applied to the extreme ends of the damaged vessel, and these portions of the artery should be excised before repair.

It is reassuring to have a pneumatic tourniquet in place proximal to the site of arterial injury during the operation in some large, distal wounds. It should not be inflated until it is actually needed. If inflation is necessary, the tourniquet should be released as soon as the bleeding vessel is under control to allow flow through the collateral vessels. Particular care should be directed toward complete deflation of the tourniquet after its use to prevent excessive venous bleeding

caused by obstruction of venous outflow from a partially deflated tourniquet.

Debridement and Evaluation of Patency

After control of bleeding from the damaged artery, debridement is accomplished by the standard technique. Before arterial repair, complete debridement of devitalized tissue, including the damaged artery, is imperative. On the other hand, debridement of the artery itself should be as conservative as possible; remove only grossly injured artery and transect the vessel at a point where the wall is grossly normal. Although microscopic changes may be found in the arterial wall in grossly normal artery adjacent to the obviously traumatized artery, resection of one or more centimeters of normal-appearing artery is unnecessary.

Distal arterial patency may be evaluated before repair by careful passage of a Fogarty balloon catheter. Distal thrombus may thus be cleared. Overzealous manipulation of the catheter, on the other hand, may inflict additional arterial trauma. Evaluation of distal arterial patency from the amount and rate of back-bleeding is unreliable. Only the reestablishment of distal pulses after repair can be considered proof of distal arterial patency. The possibility of an additional artery injury, either close to or distant from the recognized injury, must be considered and demonstrated either by intraoperative arteriography, when available, or by direct exploration of the distal artery. Failure to repair a second arterial injury usually leads to a poor result regardless of the precision of the repair of the initially recognized injury.

In laceration of the artery without transection, repair by lateral suture may be performed if the wound involves a large artery and the wounding agent has been a missile of low velocity or a sharp instrument. On the other hand, a small, high-velocity missile may create significant injury over a long segment of artery. Thrombus formation occurs both proximally and distally to the arterial wound and its presence must be suspected despite a well-transmitted pulse distal to the partial arterial laceration. If this clot is not removed and the defect

is not corrected, thrombosis or secondary hemorrhage may follow. In addition, the small missile also may have perforated the arterial wall on the opposite side, emphasizing the need for thorough mobilization and inspection of the lacerated artery.

Subadventitial hemorrhage of minor significance occasionally may give the artery the appearance of more extensive damage than actually exists. Conversely, a subintimal hematoma can occlude the artery, and an intimal tear with intramural dissection and prolapse of an intimal flap may result in thrombotic occlusion of the artery. Such lesions usually result from blunt trauma or the temporary cavitational effect of a high-velocity missile passing close to the artery.

Conservative Management

Some arterial injuries may be treated in the acute stage without an operation. When an artery is severed, there may be little or no external evidence of hemorrhage because of vessel retraction and compartmental compression from expanding hematoma. When the pressure in this area becomes equal to the pressure in the damaged artery, a pulsating hematoma develops. When both the artery and the vein are injured, an arteriovenous fistula may form with a flow of blood from the high-pressure arterial side to the low-pressure venous side. Patients with well-established arteriovenous fistulas who present no problems of secondary hemorrhage, and *whose extremities are viable*, require only low priorities for operation in combat areas. This policy is also justified for pulsating hematomas when recognition of the arterial injury has been delayed and *viability of the limb is not in question*. The patient with a pulsating hematoma must be observed carefully, nevertheless, because rapid expansion of the hematoma may compress vital structures and infection may lead to secondary hemorrhage.

When the surgical capabilities are satisfactory, there is little justification for nonoperative management of arterial injuries. Delay of operation in hopes of development of a false aneurysm or arteriovenous fistula with concomitant adequate collateral circulation cannot be rationalized except when the capability to perform arterial surgery is nonexistent or marginal.

Surgical Repair

There is no contraindication to the repair of the vessel at more than one level, but the situation should be evaluated before time is spent in repairing a damaged artery only to find another similar area within a short distance of the first.

Anastomosis.—Lateral suture repair is suitable only for a small, clean-cut laceration of a large artery (fig. 15). An autologous vein patch graft can be utilized to prevent stenosis of the repair site but ordinarily should not be utilized in a partial severance of the vessel caused by a high-velocity missile. Damage at a distance in the vessel precludes success with such a repair. If the wound is large and irregular, it is better to excise the damaged segment and perform an end-to-end anastomosis.

After the artery has been debrided adequately, noncrushing vascular clamps are applied to the vessel about 1 centimeter from each end. At this point, it is determined if the ends can be apposed without undue tension. Undue tension on the suture line may result in dehiscence and hemorrhage or may cause spasm and thrombosis. The surgeon readily develops judgment concerning the amount of tension which can be applied safely in a vascular repair.

If it is decided that too much tension exists for satisfactory anastomosis, further dissection can be carried out proximally and distally. A moderate amount of dissection frequently will compensate for a defect as large as 2 centimeters, particularly in the brachial and superficial femoral arteries. This can be accomplished frequently without sacrifice of major branches. Small branches of the damaged artery should be sacrificed only with great caution. These branches may be important contributors to collateral supply of the limb, and some of them, such as the geniculate branches, should not be sacrificed.

Direct anastomosis is performed as follows: A continuous suture is placed through the full thickness of the vessel wall about 1 millimeter apart and 1 millimeter from each cut end. In vessels the size of the radial, ulnar, or posterior tibial arteries, an interrupted suture technique should be used. Care should be taken not to allow the adventitia to be pulled through the repair site into the arterial lumen, but it is not necessary to

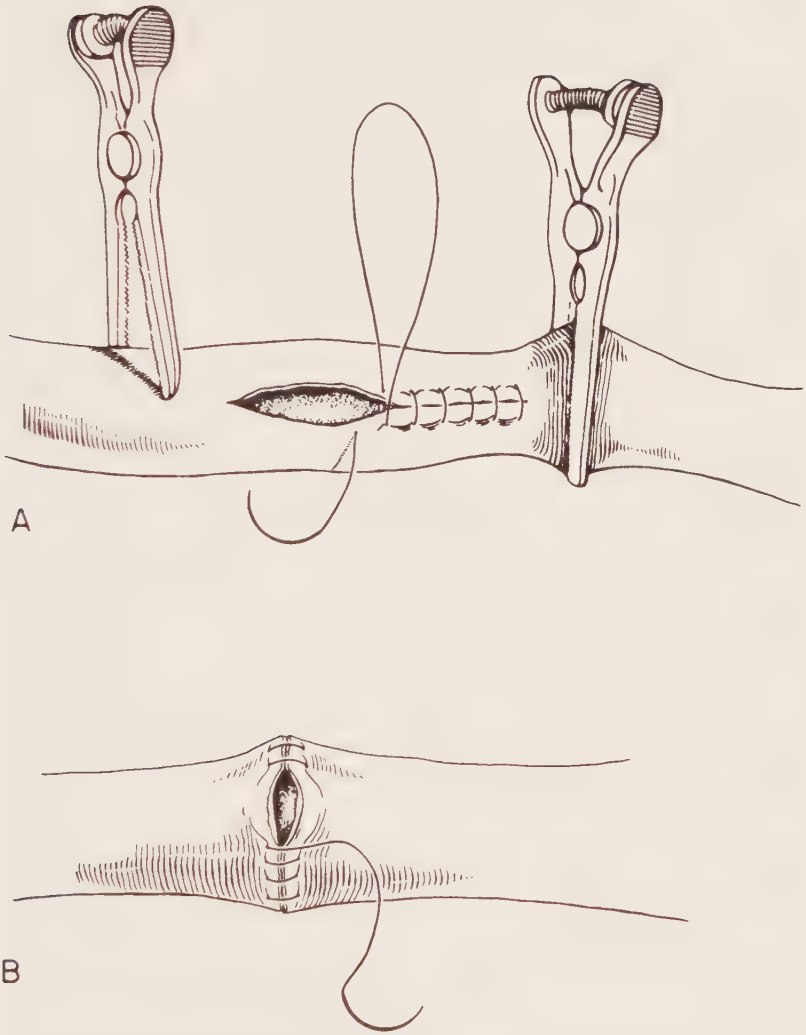


FIGURE 15.—Repair of arterial injury. A. Technique for temporary occlusion of vessel and suture repair of arterial wall. B. Technique of anastomosis.

perform an additional excision of the adventitia. To obviate distortion of the suture line with pleating, tucks, or folds, the vessel ends should be held in a constant relationship by two stay sutures as the continuous suture is applied. A simple over-and-over continuous suture with end-on coaptation joining intima to intima is satisfactory. Synthetic vascular sutures (00000 and 000000) on fine, curved, noncutting swedged needles are used. If these are not available, braided arterial silk sutures lubricated with sterile mineral oil are satisfactory.

During the repair, the lumen of the vessel should be inspected to insure that no fibrin or clots remain attached to the walls. If they are found, they should be removed by flushing the lumen with physiologic saline solution.

The tension with which the suture is held is important. The severed artery is usually in some degree of spasm, and if the continuous suture is too tight, the added constrictive effect will be undesirable. Conversely, if tension is inadequate, sutures may be too loose to prevent leakage from the suture line. Small leaks usually are controlled by pressure alone, and 5 to 10 minutes should be spent in this attempt. Surgicel (reconstituted oxidized cellulose) may be used to aid in bringing about a dry suture line. If these conservative measures fail, additional interrupted sutures may be necessary.

The anatomic location of these repairs must be considered before operation is concluded. Repairs made under normal tension in the thigh or the arm do not require the protection needed when they are made at the level of a joint. Since arterial repair at a joint usually is performed with the extremity in partial flexion, the extremity should be extended and the arterial suture line examined to ascertain that undue tension does not exist.

A bounding distal pulse sometimes is observed to return in a few seconds after the arterial repair. In other instances, the pulse may not be perceptible for 3 to 4 hours after repair, even though capillary circulation is unquestionably adequate. Decreased total blood volume, vasospasm, or decrease in body temperature may contribute to such a delay. If the distal pulse does not return immediately, additional distal arterial lesions, remaining distal thrombus, or technical failure of the repair all must be considered. It is wise, therefore, to evaluate

the distal artery with intraoperative angiography if pulses are not palpable after repair.

Grafts.—Many arterial injuries can be repaired by direct end-to-end anastomosis. However, with the increased use of high-velocity missiles, the extent of the arterial damage also is increased. After adequate debridement, if the gap between the two arterial ends is too great, a graft is required and should be utilized without hesitation. Fresh autologous vein grafts, usually the greater saphenous vein, ordinarily are utilized for this purpose. Other veins, such as the cephalic vein, also can be used, but the concomitant vein of the injured artery is usually too large to use in the repair. Its diameter immediately will expand to aneurysmal proportions under arterial pressure. If the major venous return is impaired in the lower extremity, particularly at the popliteal level, the saphenous vein graft should be secured from the opposite leg.

When venous grafts are used to repair arteries, the graft should be reversed to prevent obstruction of the blood flow by the valves. Hydrostatic dilatation of the vein graft should be carried out after excision of as much of the adventitia as can be readily accomplished.

Homologous artery-bank grafts are mentioned only to condemn their use. A high incidence of thrombosis and aneurysmal dilatation has been demonstrated in followup studies. Autologous artery grafts occasionally can be utilized successfully. The hypogastric artery is a suitable donor. The use of such grafts, however, has little place in handling acute arterial wounds.

Rarely, prostheses may be utilized in large artery wounds of the thorax and abdomen. However, routine use of synthetic material for arterial replacement in acute arterial wounds is contraindicated because of the increased risk of infection. The presence of a large foreign body presages catastrophe in most cases.

Management of the concomitant vein.—The major accompanying vein should be preserved whenever possible. Ligation is theoretically not desirable. The ligation of such a concomitant vein as the common femoral vein, for instance, may result in disabling venous stasis of the extremity. Careful lateral repair or anastomosis of the vein can be successful.

Utilization of autologous vein grafts in the venous system, particularly in the lower extremity, also should be considered. Patency for even 24 to 72 hours may be sufficient to allow the establishment of additional collateral venous return. Also, if there is an early thrombosis of the vein graft, recanalization within 4 to 6 weeks is a possibility. Increased operating time or the general condition of the patient may preclude the performance of an otherwise desirable venous repair.

Coverage of the repair.—After arterial repair, it is imperative that the affected artery be covered adequately. An exposed arterial repair will dehisce, since healing which normally progresses from surrounding viable tissue is impaired, and superimposed infection is inevitable. Interposition of viable musculofascial tissue between an arterial repair and a fracture also is indicated to obviate secondary injury or compression of the artery from bone fragments or developing callus. Muscle is the preferable covering and should be secured by transposition, if necessary. Subcutaneous tissue is a possible expedient, but bare fascia is a poor substitute. In the extreme situation, where periarterial soft tissues are massively injured, a long vein graft placed extra-anatomically in normal soft tissues may be used to reestablish arterial continuity.

Ligation.—The expected incidence of gangrene of an extremity after ligation of the more critical vessels is predicted as follows: high axillary and high brachial ligation, 45 to 56 percent; low brachial ligation, 26 percent; common femoral ligation, 81 percent; superficial femoral ligation, 55 percent; popliteal ligation, 72 to 100 percent.

Ligation, therefore, is not a desirable method of treating arterial injuries and should be reserved for arterial damage in which repair is contraindicated by reason of the poor general condition of the patient and the inadvisability of the more lengthy operation for vascular repair. A significant incidence of gangrene, therefore, is accepted when this method must be used. Occasionally, this mode of therapy becomes necessary for secondary hemorrhage in an infected wound.

Ligation of the profunda branches in the arm or the thigh, in the absence of other arterial involvement in the same extremity, does not risk the viability of the extremity, and the possibility of acute arterial insufficiency is remote. Ligation of

a single artery in the forearm or the leg is usually safe. If more than one of these vessels are injured, surgical repair should be accomplished. In recent years, such repairs have been increasingly successful.

Ligation of the internal carotid artery may be the safest procedure for patients with carotid artery injuries who have developed hemiplegia and the established clinical manifestations of a cerebrovascular accident. Reconstitution of this artery and reestablishment of arterial flow into a region of cerebral infarction may cause intracerebral hemorrhage, leading to a fatal outcome.

Associated Injuries

Bone.—Fracture frequently is associated with arterial injuries and may involve loss of bone substance. When the extremity is shortened by a fracture, tension at the anastomosis usually is not a problem. On the other hand, the fate of the anastomosis must be considered when an associated fracture is managed by traction. Whatever the technique of fracture treatment, therefore, tentative reduction should be accomplished at the time of the vascular operation to be certain that arterial length is sufficient and that the artery is not unduly stretched. If a limb is placed in traction after arterial repair, the development of symptoms and signs of arterial insufficiency with diminishing pulses is evidence of undue stretching at the repair or compression by the fracture. If readjustment of the traction does not improve the situation, evaluation of the arterial repair by angiography or operation is indicated.

If the limb is placed in a cast after operation, the cast must be bivalved to the skin for its full length to allow for postoperative swelling and to permit immediate access to the site of the repair should secondary hemorrhage occur. Splitting or monovalving the cast is inadequate.

Although unstable fractures can compromise the vascular repair, internal fixation of the fracture is contraindicated because of the increased risk of infection. External means of skeletal stabilization should be used, such as skeletal traction or *fixature externe* (see ch. XVIII).

Nerve.—Concomitant nerve injuries, more prevalent in the upper limb, may occur in association with any vascular wound. Table 7 shows associated bone, nerve, and vein injuries occurring with the wounds of specific arteries. Repair of concomitant nerve injuries is not recommended in the combat zone (see ch. XIX).

POSTOPERATIVE MANAGEMENT

After arterial surgery, the injured limb should be kept at the level of the heart, neither elevated nor dependent. If the extremity has been flexed, gradual extension over a period of several days is encouraged to avoid development of a contracture. Active muscle exercises are begun in the early postoperative period while immobilization in bed is necessary. As soon as other injuries permit, ambulation is allowed and increased rapidly as soon as delayed soft-tissue closure has been accomplished successfully.

When arterial continuity has been restored in an extremity in which the muscle tissue is of questionable viability, the patient must be observed closely for: (1) a decrease in urinary output, evidence of acute renal insufficiency; (2) increasing fever and pulse rate, evidence of wound infection; or (3) increasing pain, toxicity, confusion, fever, and increase in pulse rate, evidence of clostridial myositis. Evidence of any of the above clinical states may be indication for excision of necrotic muscle tissue or even for early amputation of a nonviable limb. Otherwise, it is safe to defer amputation and observe the patient for 4 to 5 days until a line of demarcation is established.

If fasciotomies were not performed at the time of the arterial repair, careful observation must be maintained for the development of tightness in the fascial compartments or loss of sensation in the extremity. Fasciotomies should be seriously considered at the time of arterial repair (1) for popliteal artery and vein injuries or (2) in cases in which there has been a delay greater than 6 to 8 hours between injury and repair. Therapeutic fasciotomies should be performed at the first appearance of edema which compromises circulatory or neurologic function. Fasciotomy is more often necessary in the lower extremity to

TABLE 7.—*Arterial wounds and associated injuries, Vietnam, 1965–70*

Location	Total (number)	Nerve		Vein		Bone	
		Number	Percent	Number	Percent	Number	Percent
Axillary	59	54	91.5	20	33.8	16	27.1
Brachial	283	202	71.3	54	19.0	96	33.9
Iliac	26	3	11.5	11	42.3	2	7.6
Femoral, common	46	7	15.2	18	39.1	9	19.5
Femoral, superficial	305	61	20.0	139	45.5	72	23.6
Popliteal	217	81	37.3	113	52.0	87	40.0
Total	936	408	43.5	355	37.9	282	30.1

decompress the anterior tibial or superficial and deep posterior compartments.

ADJUNCTIVE THERAPY

Antibiotic therapy.—Broad-spectrum antibiotics should be started as soon as possible after the injury and be continued throughout the operation and for at least 5 days after the operation. Intravenous administration, particularly of the initial doses, is important especially in the patient in shock who does not absorb intramuscular medication rapidly.

Anticoagulants.—Anticoagulation of the distal arterial tree is acceptable during the operation, but systemic anticoagulation should be avoided during the operation and in the postoperative period.

Arteriograms.—Preoperative arteriograms are indicated only occasionally; however, intraoperative arteriography is helpful to rule out additional arterial injuries or to outline the presence of distal thrombus.

Sympathetic block.—Preoperative sympathetic block, used as a differential diagnostic test to rule out spasm as a cause for a pulseless extremity, is a waste of time. Spasm of large vessels is not mediated through the sympathetic nervous system. In limbs which have been extremely ischemic for prolonged periods of time, however, improvement following adequate arterial repair may be slow. In such instances, repeated sympathetic blocks may be beneficial. Sympathectomy is not indicated in the postoperative period after an arterial injury and should never be used as a substitute for adequate arterial repair or reexploration of an arterial repair if occlusion is suspected.

COMPLICATIONS

When vascular reconstruction is practiced as primary surgical treatment, approximately one-third of such patients may be expected to have major complications.

Infection and hemorrhage.—Infection occurring in a wound harboring an arterial repair usually results in hemorrhage from disruption of the vascular suture line. Additional repair should

not be attempted in the infected site. Ligation of the artery is mandatory as the minimal procedure. Occasionally it may be necessary to reconstruct the arterial supply, utilizing an extra-anatomic noninfected location, to maintain extremity viability.

Thrombosis.—It may be necessary to perform a second operation in the immediate postoperative period if early thrombosis occurs and viability of the extremity is questionable. However, if viability is maintained despite thrombotic occlusion of the repair, additional operations in the combat zone should be avoided. Repeated operations under these conditions are followed by a high incidence of infection and may prejudice life as well as limb. Additional operations can be performed at an elective time if symptoms of arterial insufficiency persist. The patient, however, may develop collateral circulation to the point where he is essentially asymptomatic despite the presence of thrombosis of a major artery and probably will not require an operation to reestablish arterial patency.

Segmental occlusive spasm may occur after arterial injury. Papavarine hydrochloride, 1-percent procaine hydrochloride, or warm packs applied locally and hydrostatic dilatation of the involved segment have been employed successfully. These procedures are performed at the time of surgery.

Contracture.—The limb which is ischemic after vascular injury may develop Volkmann's ischemic contracture. This condition may be prevented if the blood supply is restored within a reasonable period of time. When circulation returns, however, the muscles tend to swell, and if fasciotomies, with adequate incisions in the skin and long incisions in the fascia, are not performed promptly, continued edema of muscle within the closed fascial compartment terminates in ischemia and necrosis. These changes are most prone to occur in the flexor compartment of the forearm and in the anterior tibial compartment of the lower extremity.

Renal failure.—When a limb which has been ischemic for many hours is revascularized or when multiple operative procedures have been performed to reestablish arterial integrity, renal failure (see ch. X) may result from absorption into the bloodstream of myoglobin and other substances from necrotic muscle tissue.

RESULTS

The overall amputation rate of approximately 50 percent after the practice of ligation of major arteries has been lowered to about 13 percent by the establishment of routine arterial repair. Injuries of the popliteal artery continue to reflect a high amputation rate of 30 percent, even after arterial repair. Although the amputation rate remains 13 percent in the results from the Vietnam conflict, the same figure as attained in the Korean conflict, many thousands of arterial wounds have been repaired in Vietnam, whereas slightly more than 300 injuries were repaired in Korea.

CHAPTER XVIII

Wounds and Injuries of Bones and Joints

Injuries of the bones and joints incurred during combat actions may be closed but are usually open. Closed (simple) injuries are treated as they might be under other conditions, except that elective surgical procedures should not be done in forward medical installations. The management of the open (compound) injuries begins exactly as for open soft-tissue injuries (see ch. XVI). The immediate objective is the prevention of infection. In the process of staged wound management by thorough debridement and delayed wound closure, the open injury can be converted to a closed injury in a high percentage of cases. The incidence of infection is unacceptable and frequently catastrophic with variations from this course of action.

GENERAL PRINCIPLES

Open injuries of bones and joints are managed according to the following general principles:

1. Initial determination of the extent of the wound and of the structures involved. In high-velocity missile wounds, tissues far removed from the actual wound tract may be damaged.

2. Generous extensile incisions. Such incisions permit complete exploration of the wound, removal of all foreign material (clothing, soil, vegetation, accessible metal fragments), and

excision of all devitalized tissue. Small, detached bone-chip fragments should be removed, but major fragments in situ should be retained. Copious irrigation of the wound is mandatory. This debridement, properly and thoroughly done, provides the basis for prevention of infection and the successful result of all future treatment, including reconstructive surgical procedures. Relaxing skin incisions or other definitive surgery has no place in this stage of treatment. Counterincisions may be used on occasion to provide adequate drainage.

3. Arthrotomy. Wounds of joints require surgical exploration.

4. Vascular repair and fasciotomy. These are the only other definitive procedures permitted at the time of initial wound surgery.

5. The wound is left open. Bleeding points are controlled but otherwise no sutures are used.

6. Nonocclusive dressing and immobilization. The wound is covered with a bulky nonocclusive dressing and the part appropriately immobilized by a plaster cast which is then bivalved.

7. Documentation. It is important to document in the medical record all observed findings, particularly vessel, nerve, or muscle damage, in addition to the more obvious skeletal injury, for the information of those providing future care.

MANAGEMENT BY FIELD MEDICAL PERSONNEL

At the point of injury, the main objective in the management of the casualty is to evacuate him rapidly to the definitive treatment center. Wounds are covered with sterile dressings. Bleeding is controlled by local compression; a tourniquet might be necessary rarely but normally should be avoided. If used, it should not be released before arrival at a definitive surgical facility. Extremities are gently alined and splinted, but no attempt is made to reduce fractures. Fluid replacement is started as indicated.

MANAGEMENT AT THE FORWARD HOSPITAL

Following resuscitation, antibiotics should be started immediately according to principles outlined in chapter XI.

A properly applied tourniquet at the time of operation is a definite aid in debridement. Attention to accepted tourniquet usage principles is mandatory. A tourniquet used during operation should be released after 1 hour. A tourniquet should be released when the surgery is completed for control of bleeding points. With the use of anesthesia as indicated, long skin incisions are made, generally in the long axis of the extremity. Such incisions lend themselves to further extension if necessary. The full extent of the wound, including the deep fascia, is opened widely, allowing complete removal of foreign material, devitalized muscle, and other tissue (see ch. XVI). Small fingernail-size detached fragments of bone should be discarded, but larger fragments, particularly those contributing to length and circumferential integrity, should be retained. Large detached fragments should be cleansed thoroughly and repositioned. The infection that is likely to ensue in an open long-bone defect is more horrendous than that which might occur as a result of retained bone fragments, not to mention the long term problems encountered in trying to fill a bone defect. The wound is thoroughly irrigated. Vascular repairs are accomplished as indicated, but nerve and tendon repairs are never done at this stage of treatment.

No attempt is made to close the wound. Relaxing incisions, pedicle flaps, or any other definitive wound approximation techniques are not indicated at this time. The wound is dressed with one layer of fine-mesh gauze and bulky fluffed gauze, avoiding packing that might occlude drainage (see ch. XVI).

FRACTURES

The following additional points are important in the management of fractures:

1. Biplane X-ray films are desirable.
2. The fracture is reduced and alined as accurately as pos-

sible, considering other limiting circumstances. Care must be taken not to disturb a vascular repair. The fracture must be repositioned if any embarrassment to distal circulation occurs. Reduction of fractures must not cause stress or tension in the wound. The primary objective is to prevent infection and assure early wound healing.

3. A metal fixation device or other means of internal fixation of fractures is contraindicated in this stage of casualty treatment, with the possible exception of hand or foot injuries. Fractures in the presence of vascular repairs are no exception. The potential infection from the introduction of foreign material is a greater threat to the vascular repair. Stability of the fracture may be gained by applying the *fixature externe*, pins and plaster (fig. 16), or other means of external immobilization. *Fixature externe* employs transverse pins above and below the fracture site connected to rigid external bars that effectively immobilize the fracture and may apply compression to the fracture site if desired.

4. A circular plaster cast is applied for immobilization of the joints above and below the fracture. It must then be immediately bivalved to the skin. The monovalved cast has no place in early treatment. Bivalving the cast for transportation and evacuation is mandatory. Plaster casts should be marked with identifying information pertinent to the underlying injury and date of cast application for the use of transportation and receiving personnel. Plaster slab splinting is inadequate.

5. Spica casts should be constructed to avoid width much beyond that of a standard litter for ease of transportation.

6. Steinmann's pins are preferable to Kirschner wires for skeletal traction because, when incorporated into a plaster cast for evacuation, they are less likely to bend. The incorporation of traction bows into the cast is unnecessary.

7. Fracture of the humerus with or without brachial artery injury is best transported in a Velpeau-type dressing strapped across the chest (fig. 17).

8. Elevation of the extremity is desirable to control swelling.

9. Frequent patient inquiry is necessary to prevent skin breakdown from cast pressure. Complaints of pain under the cast must not be ignored. Patients in spica casts are turned at

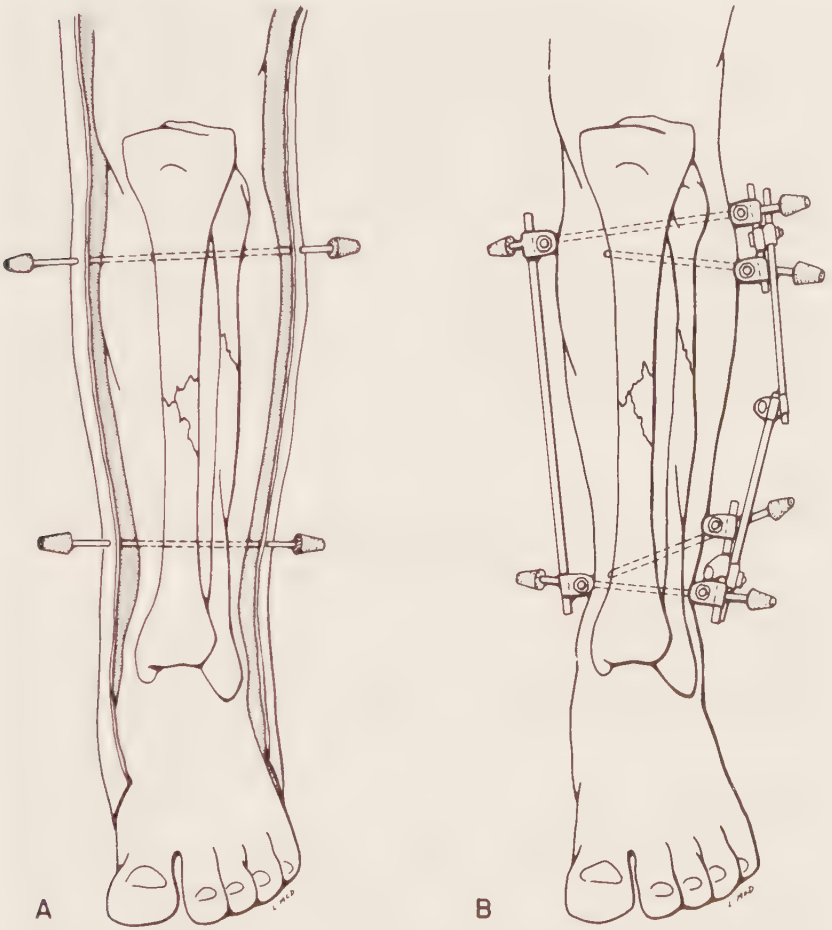


FIGURE 16.—External fixation. A. Pin and plaster technique. Cast must be bivalved. B. *Fixature externe.*

intervals to prevent pressure sores over the sacrum and other vulnerable areas.

10. The possibility of fat embolization should be considered in patients with long-bone fractures who develop signs of cerebral or pulmonary complications. Adequate oxygenation is fundamental to treatment, frequently requiring the use of a

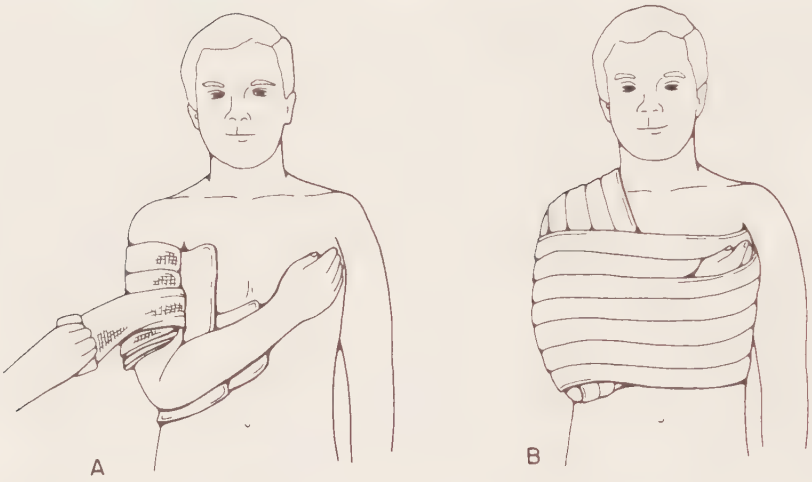


FIGURE 17.—Immobilization of upper extremity. A. Position of arm and forearm. B. Immobilization effected by binding arm to chest.

mechanical ventilator. The efficacy of intravenous alcohol or heparin is debatable.

11. Regional splinting. Preferable regional splinting is as follows:

a. Shoulder joint and humerus. A well-padded shoulder plaster spica is the method of choice. The cast is extended to include the forearm but not the wrist.

b. Elbow joint and forearm. The plaster cast is applied with the elbow in a right-angle position. It extends from the axilla over the wrist. A sling is used to support the cast and to increase the patient's comfort.

c. Wrist and hand. If the injury is limited to the wrist, the plaster cast extends from just below the elbow joint to the proximal palmar crease. The wrist is held in a position of moderate dorsiflexion with the thumb in opposition. The hand is always immobilized in the position of anatomic function and, when possible, unaffected fingers are not incorporated in the dressing.

d. Hip joint and femur. A bilateral plaster spica extends from the axilla to the toes on the affected side. The knee should not be immobilized beyond 10 to 15 degrees of flexion.

On the unaffected side, the spica extends above the knee joint. When a cast includes the foot, care should be taken that the normal arch of the foot is maintained and that the foot is not held in either inversion or eversion. When a cast includes the toes, the plaster is cut away on the dorsum of the foot to a point just proximal to the base of the toes; this precaution permits the toes to move freely and protects them from further accidental injury.

e. Knee, leg, and ankle. The plaster cast extends from the groin to the toes. The knee is immobilized as described above and the foot is at right angles to the leg.

f. Foot. A plaster cast is applied from just below the knee to include the toes as described above, with the foot at a right angle to the leg.

12. Joints not immobilized should be exercised actively.

JOINT INJURIES

A penetrating wound of a joint has a high potential for infection which can often be avoided by appropriate surgery. In addition to the described wound surgery, the following specific principles are applicable to open joint injuries:

1. Biplane X-ray films are desirable.

2. A formal arthrotomy is required. This may be done through the wound, extending the wound as necessary, or a separate standard arthrotomy incision may be required. Surgical draping should be arranged to allow movement of the joint as necessary for full exposure. Arthrotomy is to be done as early as possible in the operating room. The use of an operative tourniquet is preferred.

3. All loose bony fragments, detached or badly damaged cartilage, foreign bodies, blood, and blood clots should be removed. A comminuted patellar fracture may be treated by resection.

4. All recesses of the joint are explored.

5. The joint is irrigated thoroughly with saline.

6. If possible, the synovium or capsule is closed loosely, and the soft tissues and skin are left open. Tissue mobilization to obtain joint closure is not indicated.

7. If the capsular closure cannot be obtained, the open joint should be dressed carefully with single-layer, fine-mesh gauze. Synovium has surprising regenerative powers and often will close such defects rather rapidly.

8. Penetrating wounds of the lower abdomen and pelvic area should be evaluated carefully for involvement of the hip joint. Any evidence that the hip has been penetrated requires posterior arthrotomy and drainage. This surgery quite often will be coincident to surgical procedures of general abdominal and urologic nature.

9. Joint injuries thus treated should be dressed and immobilized as for fractures.

REDEBRIDEMENT AND WOUND CLOSURE

The wound normally is inspected in 4 to 10 days unless wound complications dictate an earlier appraisal. This inspection should be performed in the operating room at the forward hospital if the patient has not previously been evacuated. At that time, if there is significant devitalized tissue or purulent drainage, redebridement is performed. If the wound is clean, delayed primary closure is performed. The wound should not be closed with undue tension, nor with extensive development of flaps. Those wounds that cannot be closed should be deferred for later application of split-thickness skin graft. Any wound closed by the delayed primary technique should be followed carefully and reopened at any sign of inflammation or infection. If the patient is not to be retained at the forward hospital so that he can be followed for several days, it is advisable to defer delayed primary closure to those personnel in the evacuation chain who will provide proper continuing evaluation.

INFECTED BONE OR JOINT INJURIES

Modern techniques of patient retrieval and evacuation usually will provide early access to the casualty before frank infection has developed. However, if infection has already occurred,

emergency debridement is still indicated, providing wide exposure for debridement of devitalized tissue, including articular tissue, removal of foreign bodies, and release of pus. The wound is irrigated copiously, and massive systemic doses of broad-spectrum antibiotics are used. In the open infected joint following arthrotomy, debridement, and irrigation, continuous appropriate local antibiotics may be administered by intra-articular catheter perfusion. Caution must be used to avoid systemic toxicity, especially with neomycin. Five to seven days of this treatment may convert a septic joint to a clean joint. Local antibiotic solutions may be used in a similar way for infected fractures.

SPRAINS AND DISLOCATIONS

Sprains of the joints frequently are encountered in the combat zone. These injuries do not have the inherent seriousness of the missile injuries of the joints just described, but they may be severe and disabling in terms of combat effectiveness. Support of the joint by bandaging or casting will assure sound healing.

Though closed dislocations of joints are encountered less frequently, they are usually more disabling than sprains. The joint must be restored promptly to anatomical position. Reduction usually should be carried out under relaxing anesthesia unless the joint affected is a minor structure, such as a digit, or the dislocation is recurrent. Circulation always should be assessed carefully before and after reduction because associated arterial injuries may be present.

Roentgenologic evaluation is essential in all major sprains and all dislocations; it is carried out in sprains before treatment is undertaken. In dislocations, the examination is also performed after reduction to be certain that the parts have been relocated.

CHAPTER XIX

Wounds and Injuries of Peripheral Nerves

Peripheral nerve injuries, since they are not life threatening, are treated only after successful resuscitation and management of more serious injuries. Before operation, a careful neurological examination should be done. Appraisal of a nerve injury is difficult and inexact in patients who are suffering from multiple injuries, particularly in the unconscious patient. Nonetheless, the most precise clinical examination possible should be performed, with documentation of motor, sensory, and reflex findings. In the unresponsive and uncooperative patient, loss of sweating over a given peripheral nerve distribution is a very sensitive indicator of peripheral nerve injury. The experienced examiner can recognize with his fingertips the lessened friction of dry skin surface without pseudomotor function. Sophisticated tests are not necessary, but an ophthalmoscope, incorporating the +20 lens, can be a valuable instrument in the determination of sweat gland function.

CLASSIFICATION OF INJURY

The old terms "neurapraxia," "axonotmesis," and "neurotmesis" are not well understood. To simplify nomenclature, peripheral nerve injury should be described, as are other injuries to the nervous system, in the following categories:

1. Concussion (neurapraxia): a transient physiological block in nerve conduction.

2. Contusion (axonotmesis): gross evidence of damage, with swelling, ecchymosis, and loss of conduction in an otherwise intact nerve.

3. Laceration (neurotmesis): a partial or complete transection.

In addition to direct trauma, nerves can be injured by compression, stretch, or ischemia.

INITIAL WOUND CARE

Knowledge of the anatomical pathways of major peripheral nerves is essential for adequate and safe debridement of extremity wounds. The initial surgery of a peripheral nerve injury is limited to the debridement which should be performed as for any other wound of soft tissues. If the injured nerve is not seen during the course of the debridement, no search should be made to find it. If it is seen, its appearance and position are clearly recorded for future guidance. Intact nerves should be disturbed as little as possible. No attempt should be made to fix the nerve ends, nor should markers be placed at the site of damage. Nerves preferably are covered with muscle when the operation is concluded and are never left exposed in the wound. After arterial repair, the adjacent nerve should not be left directly in contact with the anastomotic site.

Primary neurorrhaphy of a divided nerve is not attempted at initial wound surgery except for digital and facial nerves. Poor results are likely to follow primary nerve suture in combat injuries for the following reasons:

1. It is impossible at this stage of the injury to determine the real extent of local nerve damage and, therefore, to determine the extent of resection.

2. Elaborate dissection necessary to mobilize a nerve trunk which must be repaired should be avoided, since this procedure extends the field of operation as well as the area of contamination.

3. Risk of wound infection which may do irreparable damage to the nerve is serious.

Some injuries will require no further surgery after the initial debridement. Partial or transient palsies, for instance, caused

by crushing and contusion of the nerve, may begin to show improvement within a few days if the contusion is slight. Only repeated observations will indicate whether or not nerve recovery can be anticipated. Improvement is heralded by a tingling sensation distal to the level of injury.

CLOSED NERVE INJURIES

Surgical exploration of closed nerve injuries is never indicated during the period of initial surgical care. These nerve injuries, like other nerve injuries, are explored as an elective procedure, preferably about the third week after injury, in a hospital equipped with facilities for neurosurgery or orthopedic surgery and physical medicine.

SPLINTING

Peripheral nerve injuries require no treatment in the division area. Splinting is desirable after emergency wound care to relax affected muscles and to prevent contractures. Pressure necrosis of insensitive skin is prevented by adequate padding of such splints and by frequently changing position.

The hand must be put into a position of function; for example, a cock-up splint is applied for wristdrop. From the very beginning, frequent active and passive motion of the joints of the digits should be carried out to avoid fixation and to preserve mobility.

In injuries of the sciatic and common peroneal nerves, the ankle should be splinted at 90 degrees with the foot in a neutral position.

CAUSALGIA

Causalgia, or aberrant pain patterns, although occurring later in the postinjury course and not usually a problem in the forward hospital, is an exceedingly painful, disabling complication of partial peripheral nerve injury. Manifesting in-

creased sympathetic nervous system activity, the patient may withdraw from all ward activities, carrying the affected part in a protective manner, and may be considered neurotic or frankly psychotic. Local infiltration anesthesia of the involved nerve, repeated sympathetic blocks, physiotherapy, and even sympathectomy have been utilized in the treatment of causalgia. The use of opiates is contraindicated, as these individuals are easily addicted.

CHAPTER XX

Amputations

Preservation of a severely traumatized limb depends upon three factors: the general condition of the patient, the extent of the injury to the extremity, and the skill and experience of the surgeon. Every effort should be made to save a limb. Conservative surgical management of injured extremities, therefore, always should be the rule. Such management includes prompt debridement, early repair of vascular injuries, prompt institution of antibiotic therapy, and postoperative immobilization. Even when the tactical situation is unfavorable, every effort should be made to arrest infection and hemorrhage without resorting to amputation. The decision to amputate requires the most serious consideration, refined judgment to determine when salvage attempts may jeopardize life, and a realistic assessment of ultimate reconstruction of a functional limb. It is always desirable to secure an opinion of a second surgeon before amputation.

Amputations for trauma are of two types, emergency and definitive. Under war conditions, all amputations performed in the forward area are of the emergency type. They are performed to save life and are done at the lowest level of viable tissue to preserve limb length. After adequate debridement of skin, muscle, and other devitalized tissues and conversion of the missile injury to a clean surgical wound, the decision to amputate the extremity or attempt to retain a viable limb frequently becomes self-evident. In upper extremity injuries, especially those involving the hand, as much viable tissue as possible should be retained for subsequent reconstruction. All attempts should be made to preserve the knee and elbow joints, even

when their preservation results in extremely short stumps. Emergency amputation is rarely the definitive surgical procedure, and further operation is usually necessary before prosthetic fitting.

INDICATIONS

The following are clear indications for emergency amputation:

1. Massive gas gangrene (clostridial myositis), the most compelling indication for amputation. Anaerobic cellulitis or myositis confined to a single muscle group can be managed by resection and is not an indication for amputation.
2. Overwhelming local infection which, despite adequate surgical measures and antibiotic therapy, endangers life.
3. Established death (gangrene) of a limb from vascular injury, when vascular repair has failed or has proved to be impractical. On the other hand, when the adequacy of the blood supply of a limb is merely doubtful, any pressure on the vascular supply to the limb should be relieved immediately. Such measures as reduction of dislocations and grossly displaced fractures or decompression of an involved fascial compartment are often effective.
4. Massive injuries in which the structures of the extremity are so badly mangled that they are obviously nonviable.
5. Secondary hemorrhage in the presence of severe infection, even though initial wound surgery apparently may have been adequate. Included in this group are patients who have not had adequate treatment early enough because of the tactical situation.
6. Extremities with severe involvement of skin, muscles, and bone with an anesthetic terminus and irreparable nerve damage.

TECHNIQUE

In all amputations, the limb should be so draped that access is circumferential to all portions of it. The limb is kept elevated during the procedure when practical, to salvage as much

as possible of the distal venous blood. A tourniquet is indicated, when practical, to prevent additional blood loss.

Site of Amputation

There are no definitive sites for amputation in the field. Amputation should be done at the lowest possible level of viable tissue. Often there is good viable skin and soft tissue distal to the indicated level of bone amputation. It is advisable to save this tissue for use in subsequent closure of the amputation stump. This is especially true in amputations below the knee in which short tibial stumps can be saved with posteriorly based flaps. Surgical principles concerning the construction and dimensions of viable flaps should be respected. A flap which is too long will not survive.

Open Circular Technique

The open circular amputation, as described below, is the most acceptable type for combat conditions:

1. A circumferential incision is made through the skin and deep fascia at the lowest viable level. This layer is allowed to retract without further dissection (fig. 18A).

2. The muscle bundles exposed then are divided circumferentially at the new level of the skin edge. The muscle bundles will retract promptly, exposing the bone beneath (fig. 18B).

3. So that the bone may be divided at a still higher level, manual pressure is applied to the proximal muscle stump, and the bone is transected, without reflection of the periosteal covering (fig. 18C). The resultant surgical wound has the appearance of an inverted cone (fig. 18D).

4. The blood vessels are divided between clamps and are ligated as they are encountered. In addition, a transfixing chromic catgut suture is added to the cuff of large arteries. The artery supplying the sciatic nerve may require separate ligation. Temporary pressure or bone wax is applied to the open medullary cavities of large bones to control oozing.

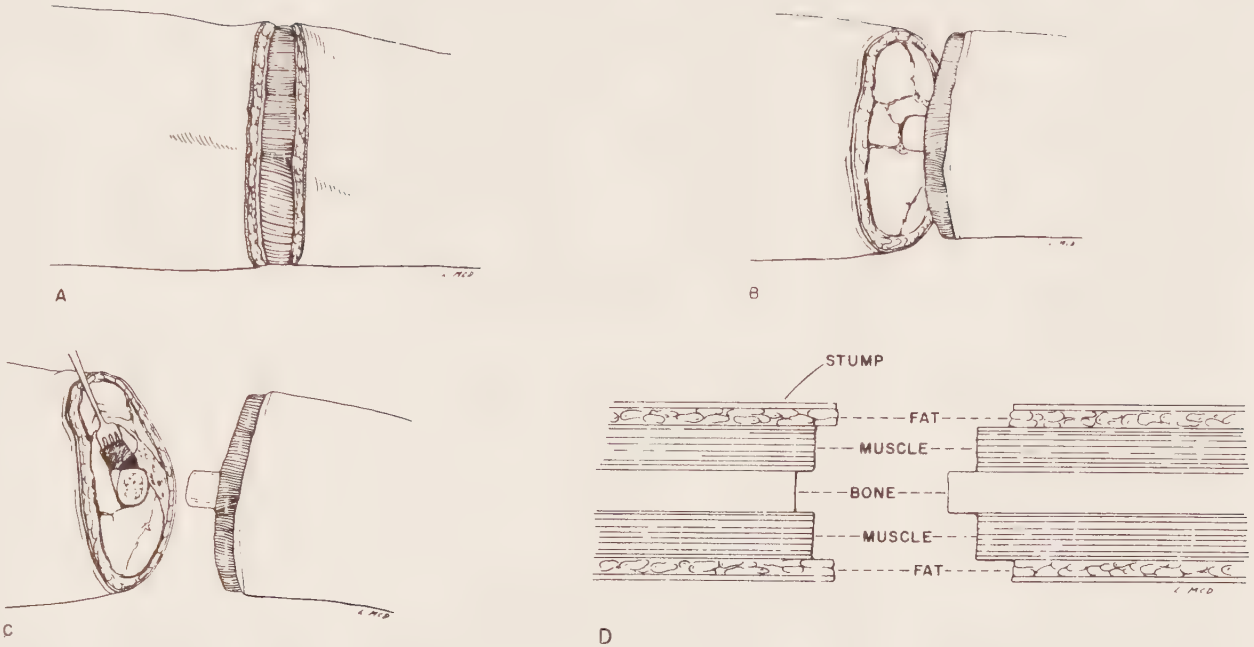


FIGURE 18.—Technique of open circular amputation. A. Circular incision of skin. B. Section of the muscles at the level of retracted skin. C. Section of the bone at the level of retracted muscles. D. The resultant surgical wound has the appearance of an inverted cone.

5. Major nerves are transected at the highest possible level without resorting to traction. Nerve stumps are neither ligated nor injected with alcohol or other chemical agents.

6. Since the amputation has been performed because of irreparable damage to a contaminated, if not grossly septic, extremity, the stump is *never* closed primarily.

Dressings

A layer of dry fine-mesh gauze is placed over the wound, and the recess of the stump is packed loosely with fluffed gauze or other suitable material. Stockinette is then applied to the skin above the open stump with a liquid adhesive to prevent slipping. The stump is wrapped with compression, decreasing proximally, and 5 to 6 pounds of traction are applied with weights and pulleys or with a self-contained traction device (fig. 19). Constrictive wrapping at or above joints must be avoided. Traction should be maintained continuously. It should be reapplied after dressing changes.

The amputation with preserved flaps requires individualized dressing consideration. The flaps should be held in their intended position by the dressing, although the major area of the amputation should be left widely open. No element of the flap should be suspended loosely within the dressing. No tacking sutures should be used. If at all possible, traction should be applied on the remaining skin elements other than the flap.

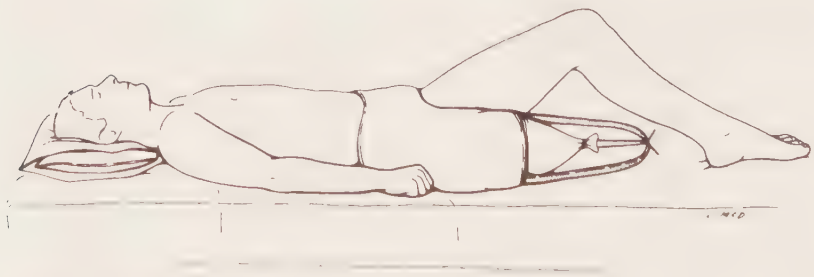


FIGURE 19.—Self-contained traction device incorporated into a plaster cast facilitates evacuation.

POSTOPERATIVE MANAGEMENT

To prevent flexion contracture of the hip after amputation through the femur, the patient should be kept in the prone position as much as possible until he has become familiar with active corrective exercises. When he lies supine, sandbags should be used to hold the stump in position. A tourniquet should be readily available for emergency use the first 5 to 7 days. It should be loosely attached to the bed or to the litter during evacuation.

STUMP WOUND CLOSURE

The timing and method of wound closure require as great a degree of surgical judgment as the performance of the original amputation. Delayed primary closure is not indicated in open circular amputations. Continued traction will often result in the skin's eventually closing over the end of the stump. If it does not, small split-thickness skin grafts can be used. A definitive revision will be necessary later, but it can be done under conditions permitting immediate fitting techniques or more rapid prosthetic application. Delayed primary closure too often results in a chronically inflamed, edematous, indurated, and sometimes draining stump that is chronically unreceptive to prosthetic application.

Where flaps have been preserved, it should be emphasized that the main basis for flap preservation is to salvage length that might be sacrificed in a rigid open circular policy. Ill-advised early closure of any amputation stump threatens length when it fails and usually results in a chronically unreceptive stump for prosthetic application.

TRANSPORTATION

Traction must be continuous throughout the evacuation process. An individualized traction device with a cast incorporating a wire ladder splint, with carefully adjusted elastic material attached to the stockinette, is sufficient. The tension

is critical, and overpull must be avoided. A self-contained traction unit may be available for transportation.

GENERAL PRINCIPLES

Certain principles in the management of amputations in the forward area merit additional emphasis:

1. Amputations are performed to save life.
2. Amputations are performed at the lowest possible level of viable tissue.
3. An obviously useless extremity should be amputated as early as possible.
4. There are no ideal levels of amputation in the forward area.
5. All amputations should be left open.
6. Early closure of an amputation is not indicated and does not hasten prosthetic fitting.
7. Cold injuries are not indications for emergency amputation.
8. Continuous traction should be provided to the amputation stump throughout the evacuation process to prevent skin retraction.

PART IV

Regional Wounds and Injuries

CHAPTER XXI

Craniocerebral Wounds and Injuries

Craniocerebral wounds are best cared for by neurosurgical teams, placed at forward hospitals. In Vietnam, a total head care team consisting of maxillofacial surgeons, ophthalmologists, oral surgeons, and neurosurgeons expanded the capability of caring for these often complex wounds.

Neurosurgical injuries are so numerous and trained neurosurgeons so limited in numbers that general surgeons are required to treat casualties with scalp wounds, some closed head injuries, and fractured skulls. Neurosurgeons are utilized to care for the more major neurosurgical problems.

CLASSIFICATION

Craniocerebral injuries can be classified into two main groups: closed injuries, without obvious external damage; and open wounds, which are further subdivided into wounds which penetrate the scalp, skull, dura, or brain itself.

Classically, injuries to the brain have been classified as concussion, contusion, and laceration.

1. *Concussion* implies a transient loss of consciousness secondary to a traumatic injury. Transient loss of consciousness is often associated with amnesia. Retrograde amnesia suggests more serious head injury.

2. *Contusion* implies anatomical and pathological change.

There is a bruising of the brain substance which may result in focal neurological impairment.

3. *Laceration* of the brain substance may result from open or closed injuries. Diffuse lacerations are often the result of severe closed injuries.

NEUROLOGICAL EVALUATION

Any examination, including the neurological examination, begins with a history whenever possible. The how, when, where, and type of missile causing the injury may be of great value to the surgeon. The neurological examination should proceed as follows:

1. State of consciousness. The state of consciousness is best described in the following terms:

a. *Conscious*—alert and aware of the external environment. This does not imply a normal mental status examination or orientation to time or place.

b. *Lethargic*—the patient prefers to sleep but can be roused. When roused, he is aware of his external environment and responds appropriately.

c. *Stuporous*—the patient prefers to sleep but can be roused. He does not, however, respond coherently to his external environment.

d. *Semicomatose*—the patient responds only to painful stimuli.

e. *Comatose*—the patient does not respond to any stimulus.

In addition to using one of the above descriptive terms for state of consciousness, the patient's actual responses to stimuli should be described in detail.

2. Evaluation of eye movements and pupillary response. Inequality and response of the pupils are very important signs in the evaluation of the head-injured patient. A unilateral progressively dilating pupil that loses its reaction to light is most significant and presages serious intracranial injury. Dilatation of both pupils is a late sign and should not be awaited. Extraocular movements may be conjugate or disconjugate and eye deviation may be very significant.

3. Motor examination. Localized weakness of one part of

the body should be noted. Included in this brief motor examination should be a recording of the results of testing of deep tendon reflexes and pathological reflexes.

4. Vital signs. Temperature, pulse, respiration, and blood pressure should be recorded. Increase in intracranial pressure may cause bradycardia and elevated blood pressure. Hypotension with other signs of shock cannot be attributed to an isolated head injury unless scalp bleeding has been excessive. Another etiology for shock must be looked for. Elevation of temperature and irregular or other pathological patterns of breathing indicate serious brain stem damage.

5. Roentgenograms. Roentgenograms of the skull are an absolute necessity. Films should be taken in two planes.

6. Recording. It is extremely important that for all head injuries the data secured as the result of initial and subsequent examinations be carefully recorded since *changing* signs are most significant in evaluation and treatment.

MANAGEMENT

All injuries must be evaluated regarding priority treatment in the rational management of the patient with a craniocerebral wound. Ordinarily, thoracic, abdominal, and vascular wounds take precedence over head wounds. The patient with a head injury, however, who rapidly deteriorates, particularly after a lucid interval, must undergo urgent operation! Restlessness in the head-injured patient, in addition to reflecting an increase in intracranial pressure, may be caused by a full bladder or hypoxemia. Hypoxemia must be corrected and is first evaluated by assessment of the integrity of the upper airway.

The airway must be cleared and continuously maintained. In the unconscious patient, this often necessitates either a tracheostomy or endotracheal intubation. When there is hemorrhage into the upper airway, a cuffed endotracheal tube is imperative. When a cuffed tube is used, intermittent relaxation of the cuff is essential if the endotracheal tube is in place for more than a few hours.

When faced with more than one patient with a head injury, priorities must be set. Moribund patients and those with

compound but not penetrating wounds are delayed. Of the penetrating wounds, those with active bleeding are operated on initially, followed by those showing progressive failure. The treatment of cerebral compression is decompression. The patient who requires the most urgent attention is often the one with a small head wound, since large gaping wounds of the skull may already be decompressed. Of those who are stable, the obtunded patient is given priority over the responsive one. Patients with suspected transventricular wounds and large herniations are usually not delayed.

Open Wounds

The head wound is inspected and the head shaved. Cautious palpation of the depth of the wound with a sterile gloved finger will often provide useful information regarding its extent.

The roentgenograms are evaluated to determine the distribution of indriven bone and metallic fragments. A metallic fragment may not be seen if its course was tangential or if it passed through the skull. If it has been retained, it may not be near the indriven bone fragments. If it is near the surface of the brain opposite the entry point, it may be associated with an intracerebral or subdural hematoma on the side of the retained fragment.

The patient is then taken to the operating room and given a general anesthetic. The patient should be positioned and draped so that wounds of exit and entry are accessible and a contralateral burr hole may be made if indicated. The operation is carried out in the following steps:

1. Scalp (fig. 20A). All devitalized and contaminated skin tissue and galea are removed with maximum tissue conservation. All foreign bodies are removed.

2. Skull (figs. 20B and C). Bone does not resist infection and a chronic infection will ensue unless debridement is adequate. Only contaminated pericranium need be removed. Pericranium should never be stripped from normal bone. A margin of normal dura should be exposed. Burr holes should be placed in intact bone adjacent to the area of damage, and bone

is then removed by rongeur towards the area of contamination.

3. Dura (fig. 20D). Only minimal trimming of the dural edges is required.

4. Brain (fig. 20E). All blood clots, devitalized brain tissue, indriven bone fragments, and visible foreign bodies are removed. Devitalized and damaged tissue is removed by gentle irrigation with physiological saline and gentle suction. A meticulous search for any embedded foreign material should be conducted, and all possible such foreign material removed. The removal of metallic foreign bodies is not so crucial as is the removal of bone fragments. If pieces of bone and necrotic brain and clot lead one to the ventricle, it also must be debrided. Precise hemostasis with electrocautery is mandatory. Through-and-through wounds of the brain are handled in the same manner. However, there is more destruction of the wound of exit than that of entry and thus more opportunity for formation of a superficial hematoma there. With this in mind, one may reduce the increased intracranial pressure more in a failing patient by operating first at the wound of exit.

5. Closure. The scalp must be closed accurately. Primary scalp closure should always be attained, even if it requires utilization of rotated skin flaps with closure of the secondary defect by split-thickness skin grafts. Closure of the dura after the brain has been debrided is essential. Closure of the dura should be attained using pericranium, temporalis fascia, galea, or fascia lata, if primary closure is not possible. Dural substitutes other than autogenous tissue should never be used in these contaminated wounds.

6. Postoperative care. Postoperative radiography should be obtained on all patients. When retained bone fragments are demonstrated and the patient's condition permits, reoperation should be performed within 48 hours. Retained bone fragments are an indication of the incompleteness of brain debridement.

Fluid and electrolyte balance must be maintained. If a patient is unconscious or unable to swallow, and abdominal injuries do not preclude it, feedings via a nasogastric tube are begun by the second or third postoperative day. The unconscious patient requires urinary drainage. Intestinal distention should be avoided and enemas may be useful in this regard.

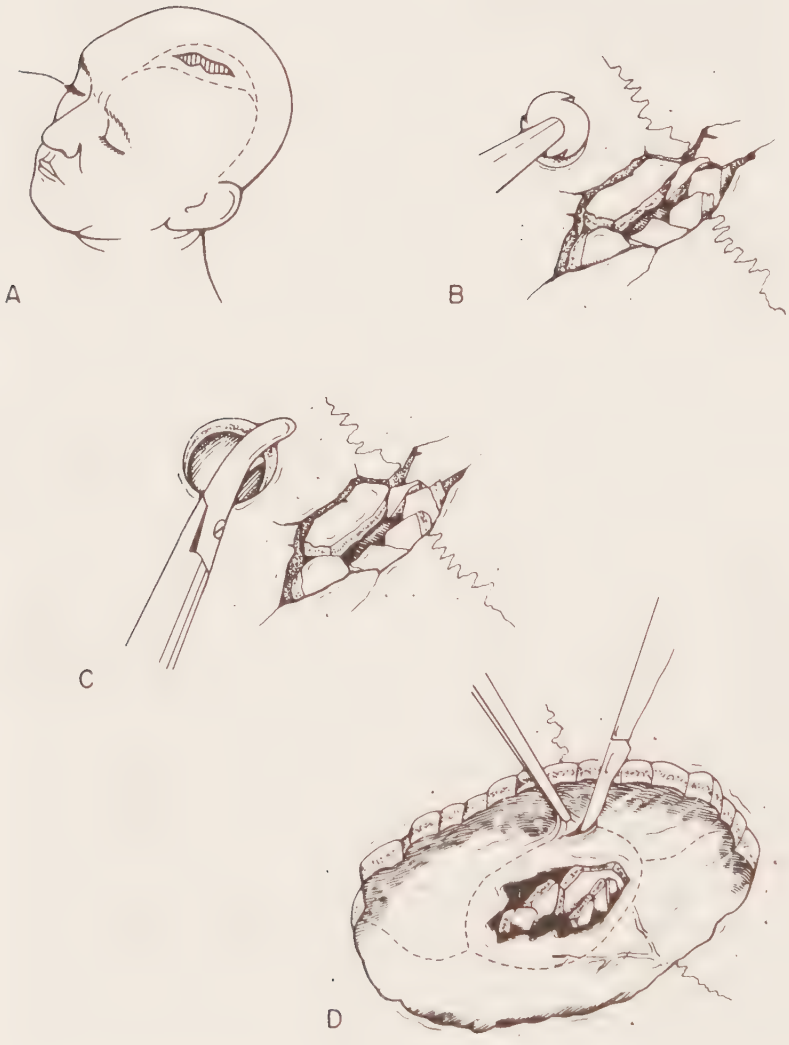


FIGURE 20.—Technique for debridement of head wounds. A. Conservative excision of devitalized skin and galea. B. Burr holes should be placed in intact bone adjacent to the area of damage. C. Bone is then removed by rongeur towards the area of contamination. D. Only minimal trimming of the dural edges is required.

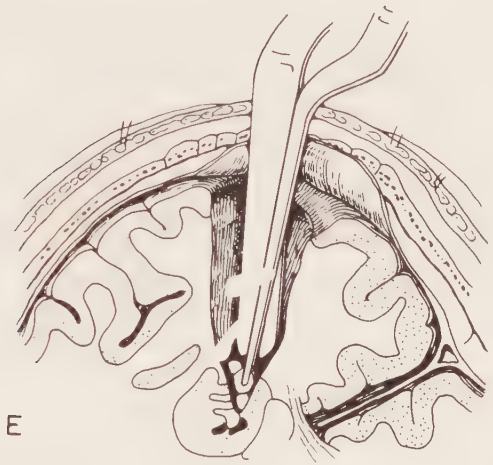


FIGURE 20.—Continued. E. All blood clots, devitalized brain tissue, indriven bone fragments, and visible foreign bodies are removed.

Most patients with head injuries do not require sedation or analgesia. When sedatives are necessary to control agitation, they should be used sparingly. Narcotics are contraindicated in the head-injured patient. Frequent changing of position is necessary to prevent skin breakdown and respiratory complications. Anticonvulsant medications are recommended. Prophylactic antibiotics are given in all cases. Steroids and osmotic diuretics may be used in selected cases to reduce cerebral edema.

Arteriography is occasionally of value in the postoperative period but seldom preoperatively in open head wounds. If in the postoperative period a cerebrospinal fluid fistula becomes apparent, it must be closed before further evacuation. Air evacuation should not be undertaken in the presence of a significant quantity of intracranial air.

Tangential Wounds

Tangential wounds of the skull require special emphasis. They may appear on inspection to be minor, but these wounds

frequently show severe dysfunction of the brain under intact coverings and should be treated accordingly. Tangential wounds secondary to high-velocity missiles always cause pathological findings out of all proportion to the apparent physical severity of injury. Compound, depressed fractures, resulting from such tangential wounds, should be treated by craniectomy rather than by elevation of the fragment. If the patient has an appropriate focal neurologic deficit or if a subdural hematoma is suspected, the dura must be opened. The underlying cortex must be debrided and bleeding controlled precisely with electrocautery. Often subdural or intracerebral hematomas will be found. If there is no evidence of bone involvement on X-ray or if only a linear fracture is present, a burr hole should still be placed for inspection of the dura.

Closed Wounds

Cerebral compression from massive intracranial hemorrhage occasionally follows closed injuries of the head. This lesion must be suspected in any patient with a head injury who develops signs of increased intracranial pressure. Dilatation of the pupils and progressive hemiplegia are late signs. Progressive loss of consciousness usually is the earliest indication of this complication.

Many patients with head injuries recover some degree of consciousness and have a lucid interval after which they progressively deteriorate. Deterioration as a result of intracranial hemorrhage is often heralded by restlessness and increasing severity of headache. Arteriography is of great benefit in identifying and localizing areas of intracranial hemorrhage. If, however, this technique is not available, trephination may be employed. In a patient whose deterioration is caused by increasing intracranial pressure, use of osmotic diuretics may be a beneficial temporizing measure.

The burr holes are made over the site indicated by neurologic signs; such as ipsilateral dilatation of the pupil and contralateral hemiplegia; bruising or hematoma of the scalp; or a fracture demonstrated by roentgenologic examination. A burr hole above the ear at the temporal crest is frequently used,

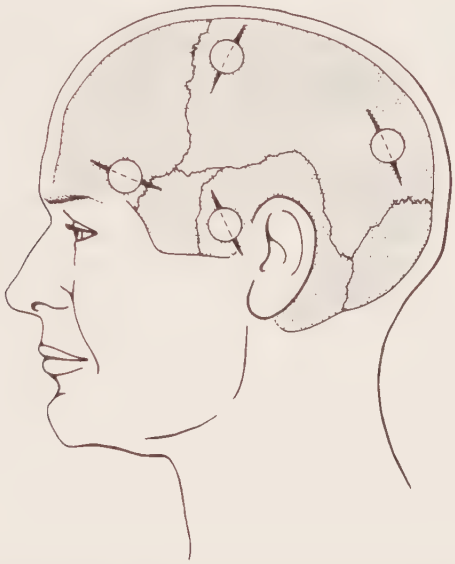


FIGURE 21.—Locations of exploratory burr holes and incisions for each.

but bilateral exploration is more often indicated. As many as four burr holes on each side are sometimes necessary before the diagnosis is established (fig. 21).

After a small incision has been made through the scalp, a skull perforator is placed in the brace, and a funnel-shaped hole is made in the skull and continued downward until the pale blue of the normal dura or the dark coloration of an intracranial clot is seen. At this stage, the perforator is replaced with burrs of various sizes, and the original opening is enlarged until a clear view of the area is possible. If necessary, the hole can be enlarged with rongeurs, or adjacent burr holes can be joined with a wire saw. When the bone has been removed, it is well to wait for a short time before proceeding with the operation, to allow the cerebral circulation to adjust itself.

Extradural hemorrhage occurs from laceration of the middle meningeal artery or from laceration of the dural sinuses. Bleeding from venous sinuses may be difficult to control and should be repaired by primary suture or by muscle tamponade. Subdural hematomas may be secondary to laceration of bridg-

ing veins or of cortical vessels. Acute subdural hematomas are often associated with significant deeper injuries. Intracerebral hematomas should be treated by cortical incision and evacuation of the hematoma rather than by needle aspiration.

EXPEDIENT MEASURES

Whenever it becomes apparent that the craniocerebral wound is of such magnitude that it cannot be handled by the skills and facilities available, the following expedient measures should be carried out:

1. Grossly devitalized and contaminated soft tissue and bone are removed, along with the foreign material visible on inspection superficial to the dura. The dura is not attacked.
2. The wound is gently irrigated with physiologic salt solution, and bleeding vessels are ligated. Gelfoam can be used to control oozing. If possible, the scalp wound is loosely approximated, to provide temporary coverage.
3. Sterile petroleum-impregnated gauze is laid over the wound, and a thick gauze dressing is held firmly in place by a bandage.
4. High priority is established for evacuation to an installation with full neurosurgical facilities.
5. The medical record is prominently marked, and attention is called on it to the incompleteness of the treatment in the forward hospital.

PROGNOSIS

The prognosis of craniocerebral injuries is good in patients who are not deeply unconscious, who respond to simple commands, and who do not develop signs of deterioration. In any head-injured patient who shows signs of deterioration, it must be determined whether or not this deterioration is due to a problem requiring surgical treatment. The prognosis is grave in patients who are rendered immediately comatose and who remain in a state of unconsciousness for a long period of time. Any improvement in the neurological condition of the acutely injured patient is significant. Restlessness and return of voluntary activity are phases which many head-injured patients go through as they recover.

CHAPTER XXII

Wounds and Injuries of the Spinal Cord

Injuries of the spinal cord are divided into the following etiologic classifications:

1. Direct injury to the cord, the nerve roots, or both may be caused by the impact of missiles or shattered bone fragments. Either crushing or transection of the cord may result. This type of injury is generally open.

2. Indirect injury to the cord may be caused by the disturbance of tissues near the spine by the passage of high-velocity missiles, as well as by other violent forces. This type of injury, which is usually closed, is of a lesser degree than direct injury. It takes the form of concussion, hemorrhage, or edema of the cord. Functional transection is also a result of such disturbances of the tissues even though the cord itself sustains no direct injury.

3. Closed injuries of the spinal cord, identical with those of civilian practice, are also encountered as the result of falls, crushing injuries, diving, and similar accidents. These injuries take the form of crush fractures and fracture dislocations. Paralysis may be of immediate onset or may be precipitated by improper movement of the patient, especially if the injury involves the cervical spine.

If there is an immediate complete loss of spinal cord function which persists for longer than 24 hours, the prognosis for significant recovery is extremely grave whether or not anatomic dis-

ruption has occurred. Laminectomy is seldom indicated in this group of cases.

Paralysis of delayed onset or gradually increasing neurological involvement may indicate hemorrhage or edema in or about the cord. A worsening neurological defect is an absolute indication for immediate operation. Hence, of inestimable value to the neurosurgeon who must make this decision is the accurate documentation of the neurological findings made by each preceding examining physician in the chain of evacuation.

EVALUATION

Neurologic examination.—If the patient is conscious, he should be asked whether paralysis occurred at the time of injury or later and whether it has improved or worsened since the onset. The head, neck, and back are inspected and palpated to identify wounds, local swellings, areas of tenderness, and other abnormalities such as gibbus, torticollis, and kyphosis. The site of pain is identified and it is determined whether it radiates down the extremities or is of the referred type.

The tone and motor power of the trunk and extremities are investigated. The extent of sensory disturbance is noted. The upper level of sensory impairment is the best guide to accurate localization of the spinal cord injury. Vibration sense and deep-pressure sense are determined and the state of the reflexes is noted. Evidence of visceral paralysis and whether ileus or retention of urine has occurred are also determined. The data obtained from this examination are compared with findings on previous neurologic examinations.

Roentgenologic examination. — Biplane roentgenograms should be obtained. A physician should be in constant attendance to supervise positioning during these maneuvers to prevent further injury. These films are essential to determine the presence of fractures or dislocations. Myelography is helpful in localizing spinal cord fluid blocks.

Lumbar puncture.—Lumbar puncture is not essential in the early management of the patient. It should be deferred until definitive surgery is considered.

MANAGEMENT PRINCIPLES

The effective management of paralysis caused by spinal injuries, whether they are closed or open, begins at the place at which the patient falls at the time of wounding and continues until his final rehabilitation. In evacuating casualties with spinal injuries from the battlefield, great care must be taken first to immobilize the spine. This may be accomplished with a litter, a wide plank, or any expedient material of suitable size. This precaution must be followed when extracting a casualty from a disabled vehicle or any other awkward situation. Motion at the site of spinal fracture or dislocation may result in transection of an intact spinal cord.

Essential principles of care of the patient with spinal cord injury are as follows:

1. The patient's condition should be discussed candidly with him. False hopes, which are unwarranted, should not be encouraged. Every patient should understand that his progress depends upon his own active interest in each step of his management. A cheerful team spirit in the staff will encourage a similar spirit in their patients.
2. Skilled and experienced nursing outweighs all other phases of treatment. Decubitus ulcers are prevented by frequent turning and careful skin hygiene.
3. Correct management of the neurogenic bladder decreases urinary infection and the possibility of calculus formation.
4. Fluid balance and good nutrition are maintained by a high fluid intake and a high-caloric diet with a large protein component.
5. Sedatives and narcotics are avoided. Addiction in these patients is a constant threat.
6. As soon as circumstances permit, prompt evacuation of the patient to a specialized center is desirable.

CLOSED WOUNDS

Laminectomy is not usually an emergency measure. Earlier, it was commonly used as an emergency measure in fracture-dislocation with paraplegia, to determine the exact nature of

the injury by direct inspection and to decompress the nerve roots. This is no longer the policy. Experience has shown that more information can be obtained from careful clinical investigations than by direct inspection.

Laminectomy should not be performed without proper preparation of the patient, proper equipment, and experienced personnel. The three indications for this operation are as follows:

1. Incomplete cord lesions in which there is a worsening neurologic deficit.
2. Complete or incomplete paraplegia with evidence of spinal canal fluid block by manometric or myelographic investigation despite a normal appearance on routine roentgenograms.
3. Depressed fractures of the laminae and injury to nerve roots, especially at the thoracolumbar junction.

OPEN WOUNDS

Following the usual preoperative preparation for any patient sustaining serious trauma, all compound injuries and those closed injuries showing progressive neurologic deficit require operation.

The patient is taken to the operating room on the Stryker or Foster frame or similar apparatus made from two litters (fig. 22). General endotracheal anesthesia is instituted with the patient in the supine position. The patient is then rotated to the prone position in preparation for operation.

The general aim of early surgical treatment of wounds in the region of the spine is the prevention of localized and of general infection, including meningeal infection. This is accomplished by debridement, which is performed at a specialized center and not in the forward area if the patient can reach such a center within 24 hours after wounding. If he cannot, debridement is carried out by the usual technique.

Complete decompression of the spinal cord or cauda equina, removing compressing bone or metallic fragments, and a maximum effort to obtain dural closure, utilizing fascia lata or

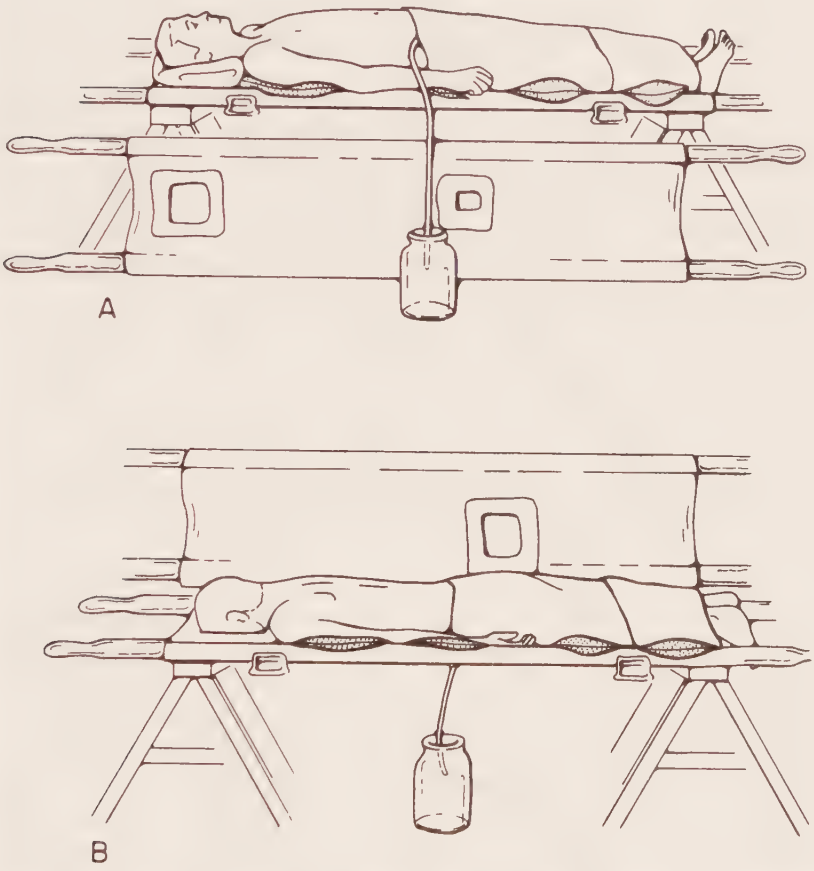


FIGURE 22.—Stryker frame improvised from standard litters. A. Patient supine. B. Patient prone.

lumbar fascia, are necessary. Special emphasis is given to isolating the spinal wound from an abdominal wound when the combination is encountered. Continued contamination of the subarachnoid space from a kidney or colon wound is a serious, often lethal, complication. Every effort, therefore, is made to close the dura and adequately treat and drain the retroperitoneal or abdominal wound.

REDUCTION AND STABILIZATION OF CERVICAL INJURIES

Skeletal traction is the treatment of choice in dislocations and fracture-dislocations of the cervical spine and should be instituted at the initial medical facility. In the transportation of cervical injuries, traction may be applied to the head by halter or a chinstrap. The patient's head is further immobilized by placing sandbags on each side of the head. After examination has been completed, skeletal traction is then applied by means of the Crutchfield calipers (fig. 23) or by using Vinke tongs. Skeletal traction is applied under local anesthesia.

Symmetrically placed small cruciate incisions are made in the shaven scalp 1 inch above and slightly behind the apex of each ear. When the diploë has been entered on each side by a small trephine, the points of the caliper are introduced and are securely fixed by a double screw-locking device. A small dressing is applied around each incision.

Traction is begun by using 15 pounds over a pulley fixed to the head of the litter or frame. The patient is then ready to be moved to a Foster or Stryker frame. Reduction is frequently obtained at once or within a few hours. If not, the weight is gradually increased in 5-pound increments at 30-minute intervals until reduction is obtained. During this period, continuous neurological examination and observation, as well as frequent lateral roentgenograms, are important. The total weight used to achieve closed reduction should not exceed 30 pounds. No matter what method of traction is used, the head of the bed is placed on blocks for purposes of countertraction. Casualties with fractures of the cervical spine are evacuated with skeletal traction in situ, the neck being immobilized by small, firm pillows on either side. The use of traction is no contraindication to regular turning of the patient.

SUPPORTIVE AND POSTOPERATIVE CARE

Intestinal Function

Injuries of the cervical and thoracic spinal cord, including spinal concussion, frequently result in paralytic ileus, abdom-

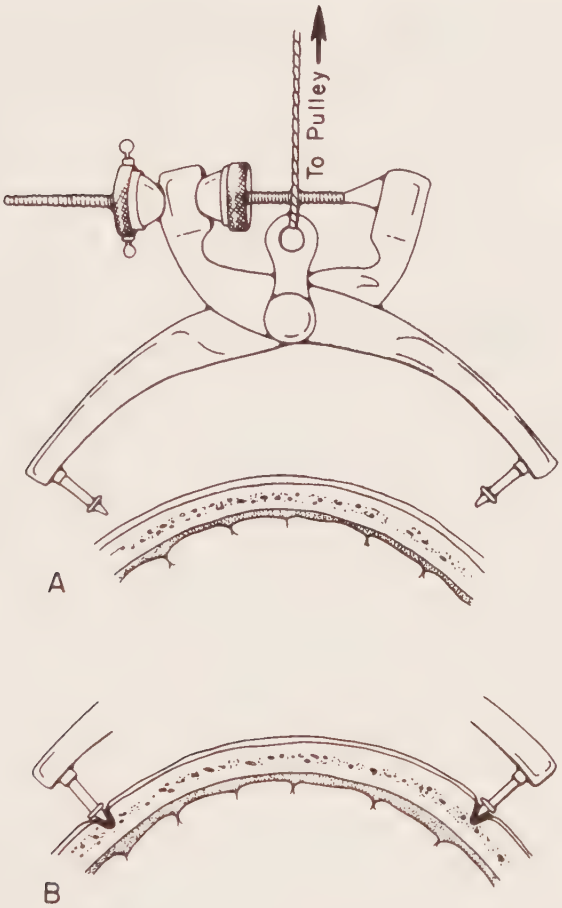


FIGURE 23.—Technique of application of Crutchfield tongs in injuries of cervical spine. A. Positioning of tongs on skull. B. Tongs in situ in proper position for effective traction.

inal distention, elevated diaphragms, and respiratory embarrassment.

When ileus is acute, oral fluids and food are discontinued. Fluids are given intravenously. A rectal tube may be inserted, and Prostigmin (neostigmine, 0.5 to 1.0 mg) is sometimes helpful. Gastrointestinal decompression with a nasogastric tube may be required. An enema is needed at least every

second day. Fecal impaction may cause diarrhea, which can lead to skin soiling and ulceration. Impacted feces must sometimes be removed digitally.

Bladder Function

Management of the paralyzed bladder due to injury of the spinal cord or nerve roots demands rigid, unremitting care from the moment of injury. The basis of management consists of preventing the paralyzed bladder from becoming overdistended. Fluid intake should be liberal to assure at least a 2,000-ml urine output. This aids in preventing calculi and infection. Suprapubic cystostomy is to be avoided unless it is absolutely mandatory. An indwelling Foley catheter no larger than 16 Fr with a 5-ml bag should be used. Antibiotics should be employed upon specific indication. A closed system of urinary drainage is necessary, and intermittent irrigation with a urinary antiseptic may be advantageous. The catheter should be taped to the abdomen to avoid urethral injury from pressure at the penile-scrotal junction.

Prevention of Decubitus Ulcers

The harm done to a paraplegic by the development of decubitus ulcers may be exceedingly serious. In addition to the hazard to life from infection and protein depletion, recovery in incomplete injuries can be retarded or prevented altogether by this complication.

Pressure necrosis begins immediately after injury. With the passage of time, weight loss is common, and bony prominences, which are sites of decubitus ulcer formation, are accentuated.

The usual nursing routine includes careful cleansing of all parts and other skin care; precautions to avoid creases in the bedclothes and contacts with hard objects which might cause pressure necrosis of the anesthetic skin; frequent inspection of the bony prominences, especially the spinous processes, sacral area, femoral trochanters, iliac spines, fibular heads, and heels. These areas are padded as necessary.

Frequent turning of the patient is essential to avoid skin necrosis from pressure, to improve the blood supply of the skin, and to promote drainage from the kidneys. This measure, which also reduces the risk of pulmonary complications, is the most important part of the care of a paraplegic until he reaches a special center.

Preparation of Double Litter

Once a patient is placed on a suitable litter (fig. 22) or spinal frame, he should not be moved from it until he reaches a hospital at which he can receive definitive care.

If a frame designed for turning the patient is not available, one can easily be constructed from two litters, which must be identical. A number of pillows or rubber pads are placed transversely on the lower litter, so arranged that nothing hard can press on the skin. An opening is cut in this litter to provide for bowel evacuation and cleansing of the perineal area. In the other (upper) litter, openings are cut for the face and for catheter drainage from the bladder.

The patient is placed on the padded lower litter, the movement being effected carefully, with the spine in neutral position. When it is time for him to be turned (every 2 hours, day and night), additional pillows are arranged transversely over the body, and the top litter is applied. The double litter, like a sandwich with the patient as the filling, is encircled with two or three straps, which are tightened to immobilize the apparatus while the turning is accomplished.

When the bottom litter is uppermost, its pillows are removed, and attention is given to the skin and to wounds of the back.

The patient, once he is placed, in it, should remain in this litter for radiographic examination, surgical treatment of the wound, and transportation.

Physical Therapy

From the onset, the patient must be made to use to the fullest the muscles which have escaped paralysis. His future ambulation depends upon their strength and development.

Deep breathing exercises and the use of accessory muscles of respiration are encouraged.

All joints of the extremities should be put through a full range of passive movements twice daily. Flexion deformity of the knees, clawing of the toes, and plantar fixation at the ankle joints must be avoided. Foot supports at 90 degrees are used when the patient is supine. When he is prone, his feet are allowed to hang downward.

CHAPTER XXIII

Maxillofacial Wounds and Injuries

The management of maxillofacial injuries is divided into three phases, as follows:

1. In the early or primary phase, the chief problems are hemorrhage, maintenance of a patent airway, the application of protective dressings, and hydration. Wounds of the face should be repaired primarily. Minimal debridement is performed and the anatomy is restored to as near normal as possible. Immediate primary repair will favorably influence the final degree and extent of permanent disability.

2. The primary concerns in the second or intermediate phase are the prevention and treatment of infection, abscess formation, sequestration, fistula formation, and delayed primary wound closure where immediate primary wound closure could not be performed.

3. In the third or reconstructive phase, the problems are plastic repair of scars and deformities, including malocclusion and the obliteration of defects by grafts and prosthetic devices. Ideally, treatment is carried out in specialized units staffed by dental, oral, and plastic surgeons who work in close cooperation with specialists in otolaryngology, ophthalmology, and neurosurgery. At least 25 percent of casualties with maxillofacial injuries also have injuries of the head and neck. In addition, dental laboratories should be available for the fabrication of dental appliances.

DIAGNOSIS

In addition to the general examination required of all casualties to be certain that wounds which are not obvious are not overlooked, patients with maxillofacial injuries require careful roentgenologic and local examination, including inspection and palpation.

Both the injured and intact sides of the head and face are examined comparatively to detect contusion, swelling, emphysema, tenderness, areas of analgesia, and distortion of bony landmarks. The examiner should look particularly for the level of the eyeballs, diplopia, periorbital hematoma, and edema which are indicative of orbital floor fracture. Otorrhea and rhinorrhea of cerebrospinal fluid origin indicate fractures involving the sphenoidal and ethmoidal bones, respectively. Temporomandibular joint function is noted, as is the integrity of the palate and buccal sulci and the alinement of the upper and lower teeth.

Wounds within the oral cavity suggest damage to the body of the mandible. The open-mouth or so-called gagging facies usually is caused by fractures of the mandibular ramus or by condylar dislocation, but it may also result from a horizontal fracture of the maxilla, displaced teeth, or hematoma formation around a posterior fragment of the mandible.

INITIAL MANAGEMENT

The problems associated with maxillofacial injuries are similar to those of other injuries; that is, control of hemorrhage, maintenance of the airway, reduction of fractures, prevention of infection, and maintenance of fluid balance. Special problems arise because of mechanical interference with breathing and swallowing. A patent airway is essential to life. Fluid and nutrition are necessary to maintain life. Both objectives are difficult to achieve in many maxillofacial injuries because of the partial or complete obstruction of the respiratory and alimentary orifices.

If patients with maxillofacial injuries require sedation, narcotics must not be used until it is certain that there is no associated cerebral injury.

Respiratory Obstruction

Respiratory obstruction in a patient with maxillofacial injuries may be due to several causes, as follows:

1. Blockage of the airway by accumulated blood and secretions or by loose objects, such as broken teeth or dentures.
2. Prolapse of the tongue, which occurs frequently with such injuries.
3. Injuries of the hyoid bone and its attached muscles, with resulting loss of control of the tongue-hyoid complex.
4. Swelling of the tongue and soft palate.
5. Laryngeal spasm, which may be caused by anesthetic agents.

No time should be lost in relieving anoxia, which can be rapidly fatal. The patient is positioned to permit drainage by gravity and the airway is rapidly cleared of blood, secretions, foreign bodies, or whatever else may be blocking it. Direct vision and strong suction are necessary.

Shock and Hemorrhage

Control of bleeding is by direct attack on the individual bleeding point. The vessel is temporarily controlled by digital pressure until permanent control can be achieved by clamping and ligation. Clamping must be done under full vision, not blindly, because there are numerous important anatomic structures in this area to which damage could be extremely serious. Ligation of the external carotid for regional control of hemorrhage is seldom necessary.

Prevention of Infection

Because of the proximity of the naso-oral passages, maxillofacial wounds are doubly exposed to bacterial contamination. The mouth, pharynx, and nose are heavily populated by a variety of pathogens which have a ready field for growth because, except for fractures of the ascending ramus of the mandible, all fractures in this region usually communicate with wounds of the mucous membrane or with the skin.

Antibiotic therapy must be begun early and maintained if serious infection is to be prevented and controlled. Oral hygiene, with particular attention to the teeth, is also necessary.

INITIAL WOUND SURGERY

The surgical field is prepared as usual, ingrained dirt being removed by gentle scrubbing with a soft brush. The eyebrows are not shaved.

Debridement.—Tissues should be handled very gently, or with fine instruments. The blood supply of the facial tissues is so adequate and resistance to infection so high that only minimal excision of the skin is necessary. From 1 to 2 mm of the wound edges are trimmed, to be certain that noncontaminated, nonbeveled edges can be accurately approximated. The trimming is done with ophthalmic scissors or a sharp No. 15 blade. The remainder of the procedure is carried out by the standard technique of debridement.

Primary wound closure.—Maxillofacial injuries furnish one of the few exceptions to the general rule that soft-tissue wounds should not be closed at the time of initial wound surgery. Primary wound closure of facial injuries is preferred to delayed primary wound closure, though this policy does not hold for associated wounds of the neck.

Ideally, treatment is carried out in specialized units staffed by dentists, oral surgeons, plastic surgeons, otolaryngologists, ophthalmologists, and neurosurgeons. In this situation, multiple system wounds of the head and neck area can often be handled under the same anesthetic by the several specialists in a coordinated team approach. Best results are obtained where there are rapid evacuation, proper hospital facilities, good equipment, improved anesthesia, and experienced surgical teams. It is also desirable to have a dental laboratory available for fabrication of dental appliances.

Closure, which must be accomplished without tension, is begun intraorally and proceeds outwardly. When there is bone destruction as well as extensive soft-tissue damage, it may be necessary to suture the buccal mucosa to the margins of the skin to cover the fracture site. Watertight closure over a

fracture is always desirable. The oral mucosa is closed with fine chromic catgut; otherwise, the finest nylon or silk, mounted on swedged needles, should be used. Skin sutures are introduced close to the cut edge and are placed not more than 3 mm apart. Temporary application of a pressure dressing may help to prevent edema and hematoma formation.

In selected cases, when a defect is so large that closure is impossible without tension or distortion, a free graft or flap may be used. All skin flaps must be carefully approximated and held in position by suturing without tension.

FRACTURE MANAGEMENT

All loose bone fragments are removed at the initial surgical procedure. Attached fragments which are grossly soiled are also removed. The remaining bone must not be left exposed but must be covered by soft tissue. A mandibular stump can be covered by suturing mucous membrane to the skin edge. If the oral cavity has not been excluded by watertight closure, the fracture site must be drained to the exterior for 2 to 5 days.

Dental problems.—Only teeth which are completely loose or fractured teeth with exposed pulp should be removed. Firmly embedded teeth are left in situ, even if they are near fracture lines. Damaged teeth are useful for immobilization of fractures. Residual molar teeth in otherwise edentulous jaws are especially valuable for fixation. Although dead, carious, or loose teeth may cause infection, they should not be disturbed at this time.

Immobilization.—Immobilization of the jaws is necessary for the early union of fractures (fig. 24). It also facilitates the healing of soft-tissue wounds, limits the spread of infection, and prevents deformity.

Several methods of immobilization of the jaws are practical, as follows:

1. The use of a preconstructed, uniform type of wire splint, with fixation by elastic traction. This is the most generally used technique.
2. The use of intermaxillary wires.
3. The use of the patient's dentures, which are fixed in

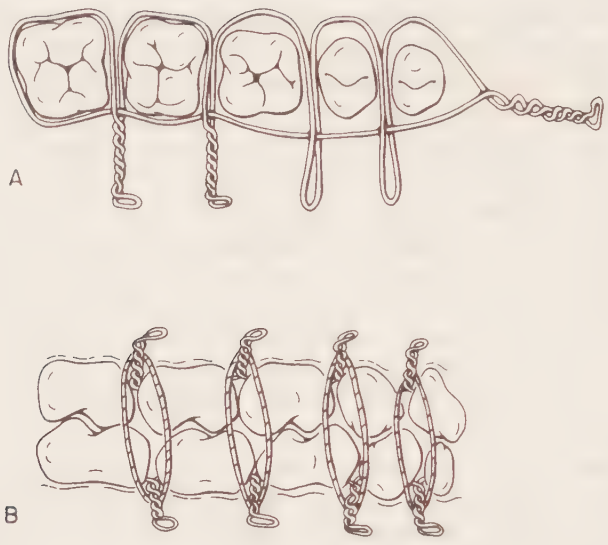


FIGURE 24.—Technique of immobilization of injured jaw by occlusive wiring. A. Application of wire. B. Occlusion by rubberband traction.

position by wires. If this method is used, one or two anterior teeth must be removed to provide for tube feeding.

4. Fixation of the lower denture to the mandible by circumferential wiring, with immobilization of the mandible to the maxilla.

5. Fixation of the upper denture to the maxilla by suspending the denture from the zygomatic arches by circumferential and transalveolar wiring.

If intermaxillary fixation is employed, some additional procedure to insure unobstructed respiration will probably be necessary.

6. The use of extraoral pins or cast metal cap splints. When there are multiple fractures and the jaws are edentulous or dentition is irregular, wiring of the fragments is satisfactory if the fractures do not communicate directly with the mouth. If they do, wiring is not satisfactory for fixation.

POSTOPERATIVE MANAGEMENT

Patients without intermaxillary fixation may be given all ordinary fluids and soft foods which require little or no chewing. If intermaxillary fixation has been used, the diet, which must be thin enough to suck through a tube, should consist of such nourishing items as milk reinforced with cream or powdered milk, lactose or glucose, thin custards, and thick soups. Feedings should be at frequent intervals and in adequate amounts.

If it is necessary to protect the lips from the wires used in intermaxillary fixation, pieces of gutta-percha are useful. Lubrication of the lips and nostrils will help to prevent fissures and ulcers.

After repair of maxillofacial wounds, a pressure dressing is applied whenever possible and left in place for at least 48 hours. Sutures are removed on the fourth or fifth day; another dressing is applied and kept in place for an additional 3 or 4 days.

REGIONAL FRACTURES

Fractures of the Mandible

A blow on the point of the chin may fracture the condyles of the mandible or the center of the body near the symphysis menti. A strong force which strikes the lateral aspect of the mandible will fracture it at the site of impact, near the canine tooth, or on the opposite side at the condylar neck. Fractures may also occur at the angle of the ascending (vertical) ramus, the condyle, and the coronoid processes. Fractures of the mandible are likely to be multiple.

Examination will reveal one or more of the following findings: restriction of the normal movements of the jaws, abnormal mobility of the jaws, an open injury extending into the mouth, irregularities in alinement of the teeth, and abnormal occlusion. Swelling and bruises of the soft palate, fauces, and lateral wall of the pharynx are seen in severe fractures of the ascending ramus.

No attempt at open reduction is made at this stage. Techniques of temporary fixation include the following:

1. Vertical bandages or elastic slings attached to a canvas headcap.
2. Interdental wire or cap splinting. This technique can be employed only when teeth are present in both fragments of the mandible and maxilla.
3. Interdental wires employed until cap splints are made. This technique can be used only when teeth are present in both fragments of the mandible.
4. Interlocking upper and lower plastic splints or circumferential wires. This technique is useful when the mandible and maxilla are edentulous.
5. Cap splints or interdental wiring for fractures of the ascending ramus.
6. Elastic traction for fractures of the condyle.

Fractures of the Facial Bones

Fractures of the bones of the face are managed according to the location and type of the injury and the degree of damage.

Fractures of the malar-maxillary complex.—Fractures of the zygomatic arch and of the malar maxilla are caused by direct violence. The malar prominence (zygoma) is flattened, although the changed appearance at first may be concealed by swelling. When the lower fracture line is low, damage to the lateral wall of the maxillary antrum must be assumed. The infraorbital nerve may be involved in the injury. If the roof of the antrum is fractured, less subconjunctival hemorrhage than usual is present. Depression of the level of the lower outer orbital margin causes diplopia. Depression of the zygoma, with impingement on the coronoid process, may interfere with opening the mouth.

Depressed fractures should be reduced at once. An elevator is introduced through an incision within the hairline over the temporal fossa, and the lever is passed between the temporal muscle and the fascia until the distal end is beneath the bony arch. The reduction is usually stable. An alternative method is to place a blunt instrument such as a urethral sound or

heavy curved hemostat under the body of the zygoma and reduce the fracture by external traction. The instrument is passed through an incision in the buccal fold near the tuberosity of the maxilla to the undersurface of the arch of the zygoma. Uncomplicated closed fractures usually can be reduced by grasping the fragments through the skin with a strong tenaculum. A useful and widely accepted method for treatment of fractures of the walls of the maxillary sinus and floor of the orbit is to enter the sinus through the canine fossa of the maxilla after the buccal mucosa over that area has been incised (Caldwell-Luc). If there is already a fracture in that area, the fragments may be removed to allow access. After entering the sinus, fragments may be repositioned into their proper place and the contents of the orbit, if herniated through the floor, returned to the orbit. The reduction is then maintained by gently packing the antrum with an antibiotic-coated gauze, a Penrose drain, or an antral balloon, which is removed in 7 to 10 days. The incision in the oral cavity heals uneventfully. Fractures involving the orbital rim may require interosseous wire fixation to maintain reduction. Again, it may be necessary to pack the maxillary antrum to maintain the level of the orbital floor.

Fractures of the nasomaxillary complex.—Direct violence may drive the upper part of the nose near the glabella inward, so that the bridge is depressed and the tip tilted upward. The upper part of the septum, as well as the nasal bones and the nasal processes of the maxilla, is involved in the injury.

If the impact is lower, separation of the nasal bones will result, and their lateral margins will overlap the nasal processes of the maxilla so that the nose appears greatly broadened. The septum may be fractured or dislocated.

Hemorrhage may be so profuse as to require intranasal packing with ribbon gauze moistened with sterile physiologic salt solution. This measure is contraindicated if cerebrospinal rhinorrhea is present.

The deformity must be corrected and the nasal airways restored without delay. Nasal forceps or blunt scissors, with the blades protected by rubber tubing, are used to reduce the septal deformity and maneuver the bones into position. When reduction is complete, the position of the bones will often be

found to be so stable that splinting is unnecessary. It is a good plan, however, to gently pack the nostrils with antibiotic-impregnated gauze to help maintain the reduced position. These packs are allowed to remain in place for 24 to 48 hours. If the reduction is very unstable, it may be necessary to use through-and-through wire sutures tied over the outside support. If stability cannot be achieved in any other way, open reduction of the nose, nasal septum, or ethmoid complex fracture may be necessary in the first 48 to 72 hours.

Associated open wounds of the nose are treated by debridement followed by primary closure. It may be necessary to undermine the adjoining skin to effect apposition without tension. If skin loss is considerable, mucocutaneous suture will be necessary to obtain soft-tissue coverage of exposed bones. Distortion of the structures must be guarded against.

Bilateral nasomaxillary fractures.—In major nasomaxillary fractures, the maxilla is displaced backward en bloc between the malar bones. The depressed, so-called dish-face deformity which results may be concealed by the extreme swelling. Many patients with such fractures also have sustained damage to the eyes, ears, and brain; and cerebrospinal fluid leaks and diplopia may be present. Malocclusion also results. The lower border of the maxilla may be rotated so far upward that only the back (molar) teeth can meet.

Patients with cerebrospinal fluid leaks should be transferred immediately to a neurosurgical unit. The risk of infection of the meninges is serious because of associated injuries in the region of the ethmoid and in the floor of the anterior skull fossa. Antibiotics are begun at once.

Impacted fractures can be reduced readily within the first few days after injury, but after 10 days, reduction is difficult and is not without danger.

The nasal airway is restored first. The maxillary alveolus then is grasped with strong, sharp-toothed forceps and is rocked, if necessary, to disimpact the displaced fragments. The maxillary fragment is immobilized in correct occlusion with the mandible. The fracture of the maxilla is further immobilized and stabilized by passing loops of wire from the mandibular interdental wiring around the zygoma. If the zygoma is not stable, the wire is carried behind the zygoma to the lateral

superior orbital rim, and skeletal fixation of the wire is effected through a small drill hole. Pullout wires are utilized to facilitate subsequent removal. Sometimes it is necessary to employ forward traction to maintain reduction. This is accomplished by elastic traction to the mandible and external connection to a plaster skullcap.

Displaced and mobile fragments present in open maxillary wounds are handled as if the injuries were closed fractures, with conservation of all bone.

Fracture of the palate.—Palatal-alveolar fractures may involve the maxillary antrum and the nasal floor. Replacement and fixation are accomplished by interdental splinting or by cap splints.

In horizontal fractures with separation of the palate (Guérin fractures), the palate is driven backward and malocclusion occurs. Reduction is effected by the technique described for major bilateral nasomaxillary fractures.

In midline (vertical) fractures of the palate, the upper incisors are separated. Reduction is by manual pressure with contact of the fragments maintained by interdental elastic bands or by cap splinting.

Fractures of the Paranasal Sinuses

Frontal sinuses.—Mild injuries of the frontal sinus may merely produce fractures of the anterior and posterior walls without displacement, for which no special care is required. Depressed fractures of the anterior wall of the frontal sinuses require cosmetic as well as functional restoration. There may be associated disruption of the nasofrontal ducts. These fractures should be repaired by open reduction and direct interosseous wiring when possible. The sinus is approached through the open wound, when present, or through an eyebrow incision in the closed injury. With direct wiring, internal support is not required. It is wise to leave a drainage tube in the sinus; the tube is brought out through the eyebrow incision. Most of these fractures are associated with some trauma to the nasofrontal duct, and the swelling and edema predispose to the development of aerosinusitis and later purulent sinusitis. The

presence of the drainage tube prevents the development of vacuum sinusitis.

If marked comminution of the anterior wall of the frontal sinus is present, it may be necessary to pack the sinus, preferably with rubber packing such as Penrose drain, until there is some fixation (approximately 1 week). The packing may be brought out through the nasal fossa or through the eyebrow incision. When the posterior wall of the frontal sinus is damaged and the dura is torn, rhinorrhea may occur. Patients with such injury should have neurosurgical consultation.

Severe injuries to the frontal sinus will produce disruption of the posterior wall and usually the roof of the interorbital space. These patients should always have neurosurgical consultation. When there is disruption of a nasofrontal duct leaving the frontal sinus without any connection to the nasal fossa, treatment must effect either reconstruction of the duct or, preferably, obliteration of the sinus. Obliteration of the sinus with an abdominal fat graft may sometimes be necessary. Inability to repair the nasofrontal duct or the presence of a cerebrospinal leak through the posterior wall of the sinus is an indication for obliteration. This procedure requires complete removal of all sinus mucosa and loose bone fragments, especially from the posterior wall. Combat injuries of the frontal sinus are frequently associated with intracranial damage, and the neurosurgical technique may involve obliteration of the frontal sinus from an intracranial approach.

Ethmoidal sinuses.—Ethmoidal sinus wounds may require a partial ethmoidectomy. If they are associated with rhinorrhea, they require observation and antibiotics or neurosurgical intervention. In severe fractures, there may be lateral displacement of the inner canthi with eye muscle imbalances which will require open reduction for correction.

Maxillary sinuses.—Simple effusion of blood into the antrum is best left alone because it is usually absorbed. If infection develops, antral puncture and lavage are employed. All patients with suspected injuries to the paranasal sinuses should be cautioned against blowing the nose. This is particularly applicable to patients with rhinorrhea because of the risk of establishing a secondary meningitis. Missile wounds involving the maxillary sinus are treated with debridement and irrigation

of the sinus either through the missile tract or through a standard Caldwell-Luc approach with intranasal antrostomy. By this method, reduction of fractures, including orbital floor fractures, is accomplished manually and held in the reduced position by packing with antibiotic-coated gauze, Penrose rubber drains, or an antral balloon. Open reduction with direct wiring occasionally is necessary. The nasal antrostomy serves as a drain site for these midface wounds. Sphenoid sinus wounds require sphenoidotomy. Often ethmoid, sphenoid, or maxillary wounds may involve orbital or intracranial contents and a combined approach is required.

EVACUATION

As little as possible should be done in forward areas for patients with maxillofacial injuries. Instead, they should be evacuated as promptly as possible to specialized facilities. Patients with maxillofacial injuries usually travel well by air. Most mandibular fractures are painful and make swallowing difficult. For transportation, therefore, these patients should be placed in the semiprone position on the litter. If there are upper respiratory difficulties, or if they are likely to develop during transportation, tracheostomy should be performed before evacuation. If tracheostomy is not performed, a patient with a maxillofacial injury must be evacuated with an attendant especially instructed in the possibilities of respiratory obstruction and in techniques of dealing with it.

Patients with major maxillofacial wounds require special preparation before evacuation to the intermediate or reconstructive care facility. If possible, the patient's infection should be under control, no significant fever should be present, and his general condition should be sufficiently stable to withstand the evacuation. Ideally, the patient should not require intravenous fluids. All packing should be removed before evacuation or specific instructions should accompany the patient concerning location, number, and types of packs with recommendations for time of removal. If intermaxillary fixation has been utilized, the patient should be retained for several days

after surgery, adjusted to a liquid diet, and tolerating fixation well before evacuation. If intermaxillary elastics are utilized, some type of pullout cords is indicated. In the patient with a tracheostomy or with missing anterior teeth, there is little likelihood of aspiration of emesis; therefore, any type of suitable fixation is acceptable.

CHAPTER XXIV

Wounds and Injuries of the Eye

Under battlefield conditions, the casualty with an injured eye is usually first seen by nonspecialized personnel of a facility in which there is little or no specialized equipment. If his injury is minor, he is treated and sent back to his unit. If it is not, he is promptly evacuated. The distinction between ocular injuries which are minor and injuries which are serious is not always easy to make. The most trivial-appearing injury may prove to be very serious indeed. If an injury of the eye is properly managed, a good result, or at least some salvage of vision, is often secured even in serious injuries. If an injury is improperly managed, a trival wound may be converted into a serious one; and a large majority of penetrating ocular injuries will result in blindness. Inexperienced personnel should refrain from interference in any injury of the eye that is not clearly minor. This cannot be too strongly emphasized! Without special instruments, without an intimate knowledge of specialized techniques, and without a sound comprehension of what constitutes a dangerous or a hopelessly injured eye, attempts at interference by untrained personnel can easily lead to disaster. Such disasters cannot be remedied by subsequent surgery. In ophthalmic surgery, the first chance at repair of an injury is usually the last. There is an inordinately high rate of ocular injury relative to the amount of surface area exposed to injury. Although comprising as little as 0.27 percent of total body surface and only 0.10 percent of the erect

frontal silhouette, the eye is injured in nearly 10 percent of nonfatal casualties. The likelihood of ocular injury is further enhanced by various postures assumed in warfare. For example, although only 25 percent of the projected body surface is exposed in the prone position, the eye comprises a considerable portion of the prone silhouette.

EXAMINATION AND DIAGNOSIS

If the patient can communicate and if combat conditions permit, an ocular examination should always begin by recording the circumstances of injury and the type of wounding agent. A penetrating ocular injury should be suspected in every wound of the eye and of the upper portion of the face until it is proved not to exist. The preliminary examination, after loose foreign matter has been flushed out of the conjunctival sac with copious irrigations of plain water or physiologic salt solution, should be conducted with the lids retracted. Since voluntary opening of the eyelids is often impossible, topical anesthesia (proparacaine hydrochloride 0.5 percent) and *gentle* lid separation with Desmarres retractors may be required for both vision testing and inspection. In the absence of lid retractors, fingers should be braced against the bony orbital rims before attempting digital separation of the eyelids. There must be no pressure at all on the globe. The slightest pressure on a globe which has been lacerated or perforated may cause irretrievable loss of the vital contents.

Visual acuity, the most important parameter in evaluating the seriousness of an eye injury, should be recorded as follows: no light perception, light perception, perceives hand motions, counts fingers, or reads. In evaluating light perception, it is important to pass a very bright light alternately in front of and away from the eye. At the same time, the other eye must be completely shielded, and the patient must be questioned carefully to detect inaccurate responses. Spurious perception of light may result simply from the patient's natural desire to see, from an awareness of heat from the light, from a sensation of air movement on the skin produced by motion of the light source, or from incomplete shielding of the other eye. The

other tests of visual acuity should be utilized with as much precision as circumstances permit.

Inspection of the eye may reveal pupillary irregularities, blood within the anterior chamber (hyphema), or even collapse of the anterior chamber, where loss of aqueous humor causes the iris to impinge directly against the posterior surface of the cornea. Lacerations of the eyelids, cornea, or sclera, foreign bodies within the eye or orbit, or disruption of the globe may be present. Gross contamination by dirt or other particulate matter frequently accompanies these injuries.

Corneal lacerations are usually evident by loss of the anterior chamber and distortion of the pupil. Iris incarceration or prolapse through the wound is common. Scleral lacerations often exhibit extruding, darkly pigmented choroid. However, small perforating wounds and even large scleral lacerations may be obscured by subconjunctival hemorrhage. More extensive prolapse of intraocular contents (vitreous humor, uvea, even lens and retina) may present within the lips of any laceration of the globe.

If the general appearance of the eye is undistorted and careful inspection reveals no site of ocular penetration, gross differences in intraocular tension may be estimated by *very gentle* digital palpation. The tips of the index fingers are used in ballottement of the globes through the upper, closed eyelids. First, test the tone of the unaffected eye, then compare with that of the injured eye. Asymmetric tension is indicative of serious ocular injury to the softer eye.

Unless total disruption is evident, the possibility of salvaging the eye should be considered. This possibility exists even in the face of questionable light perception since vitreous hemorrhage alone may mask perception of light.

MANAGEMENT

Minor Injuries

Minor ocular injuries which may be handled safely in the division area include hemorrhage of the eyelids, subconjunctival hemorrhage, superficial foreign bodies, and corneal abrasions.

Irrigation of the eyes and removal of superficial corneal foreign bodies may be performed under 0.5-percent Ophthaine or Ophthetic (proparacaine hydrochloride 0.5 percent) or 0.5-percent Pontocaine (tetracaine hydrochloride) analgesia. Some sharp-pointed instrument, such as a large needle or eye spud, should be used. The superficial abrasion left after the object is removed is treated by instillation of an antibiotic ointment and patching. If the particles are found to be multiple and more deeply embedded than has been anticipated, the patient should be evacuated.

Subconjunctival hemorrhages associated with neither decrease of visual acuity nor with blood in the anterior chamber (hyphema) or in the vitreous humor require no treatment. However, thorough ophthalmoscopy through a well-dilated pupil is necessary before returning the patient to duty. If blood is found in the anterior chamber or the vitreous, the patient should be placed on bed rest, with elevation of the head, binocular patches applied, and prepared for immediate evacuation.

Contusions of the eyelids and eyeball should likewise be examined carefully. If there is only subcutaneous and subconjunctival hemorrhage, without intraocular hemorrhage or disturbance of vision, the patient can be returned to duty.

Foreign body sensation, aggravated by blinking, and pain referred to the upper lid are characteristically found with corneal abrasion, which is usually minor, but always a painful lesion. Documentation by use of a fluorescein strip, placed momentarily in the conjunctival fornix, may be desirable. The abrasion can often be seen merely by focusing on the anterior corneal surface with +8D to +12D lenses (black numbers) in the conventional, direct ophthalmoscope.

The treatment of ordinary corneal abrasion consists of: (1) cycloplegia, using two drops of either scopolamine hydrobromide 0.25–0.5 percent, cyclopentolate hydrochloride 1–2 percent, or homatropine hydrobromide 5 percent; (2) instillation of ophthalmic antibiotic solution or ointment; and (3) application of a tight patch to insure immobility of the eyelid. The patch can usually be discontinued in 24 to 36 hours, but repatching for another 24 to 36 hours may be necessary for larger abrasions. Lack of progressive improvement demands

referral to an ophthalmologist. The use of topical anesthesia for other than facilitating vision testing, examination, or instrumentation is contraindicated. Repeated instillation inhibits healing. Topical steroids or steroid-antibiotic combinations are likewise contraindicated. Steroids are unnecessary and will cause rapid progression of a dendritic ulcer, including corneal perforation, should this lesion exist or supervene. Fungal superinfection and glaucoma may also result from injudicious use of topical steroids.

Major Injuries

Division area.—The management of ophthalmic injury begins at the battlefield or aid station. Only first aid is administered in these forward areas, and all significant casualties are evacuated to facilities where a physician is assigned. Early identification of ocular injury is urgent. In severe injury to the globe, inadvertent delay in ophthalmologic care can mean the difference between salvage and loss of the eye.

Any abnormality in the appearance of an eye injured by blast or fragmentation weapons, or by severe blunt trauma, demands the following course of action preparatory to evacuation:

1. Instruct the patient *not* to squeeze his eyelids.
2. Do not remove any penetrating foreign body protruding from the globe, as ocular content may be extruded.
3. Occlude both eyes, but avoid any pressure directly on the eyes. A battle dressing tied around the head suffices.
4. Give systemic analgesics for moderate to severe pain.
5. Evacuate immediately as a supine litter patient to a forward hospital, preferably with ophthalmology capability.

Ocular burns are usually first seen in the division area. Ultraviolet, thermal, and nonalkali chemical burns are treated as for corneal abrasions. However, nonalkali chemical burns require initial irrigation with tapwater or saline solution for 10 to 15 minutes under topical anesthesia.

In white phosphorus burns of the eye, instillation of 5-percent copper sulfate solution identifies particles, which are otherwise presumptively located by foci of smoke, by darkening them. Larger particles may require removal with a needle or spud.

These patients urgently require treatment by an ophthalmologist. In his hands, continuous irrigation with ophthalmic antibiotics in Ringer's solution may be performed via a percutaneous, indwelling, superior fornix angiocatheter since severe edema of the lids often prevents the conventional administration of topical medication. Alkali burns may result from exposure to sodium hydroxide, lye, quicklime, ammonia, and agents often found in degreasing solvents. Such a burn is an ocular emergency! Chemical penetration is so rapid that irrigation with copious volumes of water or sterile saline must be initiated within seconds. This irrigation must be continuous for at least 60 minutes. Irrigation should be continued until pH remains below 8.0 for at least 5 minutes after irrigation ceases. An alkali burn is potentially devastating and prognosis may be poor, especially if the cornea appears cloudy or the conjunctiva blanched. Atropine sulfate 1 percent and steroid-antibiotic ointments (dexamethasone sodium phosphate and neomycin sulfate) are definitely indicated here and should be instilled in the conjunctival cul-de-sac four or five times daily. Prompt evacuation is necessary.

Forward hospital.—In the absence of an ophthalmologist, treatment of major ocular injuries in forward hospitals normally is managed by the general surgeon and ideally is limited to interim measures aimed at prevention of infection within the eye. Systemic antibiotics and tetanus prophylaxis should be instituted at the earliest opportunity in the preoperative period.

Lid and conjunctival debris should be carefully irrigated away. Any sterile irrigating solution, including water, is acceptable. This should be followed by generous topical application of fresh solutions of an opththalmic antibiotic (gentamicin sulfate, chloramphenicol, or neomycin sulfate-polymyxin B sulfate) and atropine sulfate 1 to 4 percent.

A sterile, 4- by 4-inch gauze strip is applied to keep the area clean, and additional protection is afforded by taping a Fox (or similar type) shield over the injured eye. A pressure dressing should be avoided as it may cause serious damage by expressing intraocular contents through a penetrating wound. Since patching also helps provide an excellent culture medium for bacteria, particularly *Pseudomonas* species, topical antibiotic

solution is carefully reinstilled every 4 hours, and a fresh, sterile gauze reapplied twice daily. Sterile irrigation of mucopurulent secretions from the lid margins and conjunctiva should be carried out when the gauze dressing is changed. The uninjured eye should be patched to reduce unwanted ocular motion.

No ocular surgery should be performed. Particularly, no attempt should be made to remove protruding or penetrating foreign bodies or to repair corneoscleral lacerations. Preferably, neither should repair be undertaken for lacerations involving the lid margin or the nasolacrimal apparatus. Even an eye which appears grossly irreparable may have surgery deferred, utilizing the same regimen of sterile gauze dressings and antibiotics.

Until recently, the selection of systemic antibiotics has been beset with two problems: (1) many drugs do not pass the blood-aqueous and blood-retina barriers to give adequate intraocular tissue concentrations; and (2) earlier drugs have had limited bactericidal spectra, especially for strains of *Pseudomonas aeruginosa*. When ophthalmologic care will be delayed, the best initial choices of parenteral antibiotic are:

1. Cephaloridine 1.0 g I.V. q6h or
2. Methicillin 2.0 g I.V. q8h and probenecid 0.5 g q6h P.O. (or per rectum) combined with
   a. Chloramphenicol 1.0 g I.V. q8h or
   b. Ampicillin 1.0 g I.V. q6h.

While ideally handled by an ophthalmologist, many of the following ocular injuries can be managed by other physicians:

1. Eyelid laceration, with and without margin involvement.
2. Deeply embedded corneal foreign bodies.
3. Ocular burns.
4. Ocular contusion injuries.

If evacuation or ophthalmologic care is delayed, repair of lid lacerations by a nonophthalmic surgeon may be necessary. Repair should be attempted only if fine instruments and suture material are available and if the following tenets of plastic surgical technique are observed:

1. Copious irrigation of the wound.
2. Minimal debridement.
3. Anatomic reapproximation of the lid margin in two layers:

a. An initial 5–0 braided suture between the lash line and the mucocutaneous junction of the lid margin (the gray line).

b. Interrupted 6–0 or 5–0 chromic catgut for the conjunctivotarsal plate, burying knots, if approach is via everted lid.

c. Interrupted 7–0 or 6–0 silk, using small bites, for the skin and at the mucocutaneous junction.

4. If tissue loss is so extensive as to expose the cornea, temporary cover is provided by approximating whatever tissue is available. A layer of ophthalmic antibiotic ointment is maintained continuously over any residual exposed cornea. Thin, clear plastic sheeting or similar material, secured simply by ointment to periorbital skin, provides an effective moisture chamber. Priority evacuation is then arranged.

Under no circumstances should an eye be excised by a general surgeon in a forward unit unless the globe is completely disorganized by the extent of the damage or its removal is necessary as part of the general surgical management of an extensive wound of the face.

In the unlikely circumstance that a patient with a severe ocular injury cannot be evacuated within several days to a facility that has an ophthalmologist, primary enucleation should be considered if the patient has no light perception using the brightest available light source. Such a severe injury would most likely be an extensive corneoscleral laceration with either prolapse or loss of intraocular contents.

This policy of delay is perfectly safe as it relates to sympathetic ophthalmia. Sympathetic ophthalmia (involvement of the uninjured eye) never develops until at least 10 days after trauma and only very exceptionally develops before 21 days. There is sufficient time for the patient to reach an ophthalmologist.

If the decision is made to remove the eye, the conjunctiva is incised at the limbus to separate it from the globe. Using a combination of blunt and sharp dissection, the four rectus muscles are exposed from their insertions as far posteriorly as possible (usually 10 to 15 mm). Tenon's capsule (the connective tissue surrounding the globe) is separated from the globe in the four quadrants between the rectus muscles. The rectus muscles are then severed from the globe. The two oblique muscles are severed from the globe at this time.

Traction should be exerted on the globe in the anterior direction as a curved Halsted clamp is placed behind the globe as deeply into the orbit as possible. By blunt dissection, the optic nerve is isolated, clamped to crush the central retinal vessels, and cut distal to the clamp. The globe is removed from the orbit. After the Halsted clamp is removed, hemostasis should be achieved by temporary tamponade. If available, it is most important to place a glass sphere (usually about 18-mm diameter) in the position occupied previously by the globe. If a glass or plastic sphere is not available, the closure should nonetheless be completed.

Probably the most important step in the operation is the meticulous closure vertically or horizontally of Tenon's capsule with interrupted 4–0 chromic catgut sutures. The conjunctiva is closed horizontally with interrupted 5–0 plain catgut sutures. If available, a conformer should be placed between the bulbar and palpebral conjunctiva to prevent obliteration of the conjunctival cul-de-sacs and difficulty in the patient's subsequent wearing of a conformer.

In the event that the patient still retains light perception or even better vision in the face of a corneal or scleral laceration, primary closure of the wound should be performed by the nonophthalmologist physician if the patient cannot be treated by an ophthalmologist within a few days. The guiding principle is meticulous wound closure without debridement, except for the excision of prolapsed intraocular tissue.

Magnification of any type will be of great assistance. Instruments should not be introduced through the wound into the eye. If the laceration involves both the cornea and sclera, the cornea should be repaired first. The smallest (7–0) silk suture material available and the finest available instruments should be used. The first suture should not be placed until the edges of the wound are carefully alined. Close attention to the limbal landmarks will assist in proper alinement. The curved needle is introduced almost perpendicularly into the tissue about 2 mm from the wound edge and is taken to midstromal depth (the cornea is less than 1 mm thick in most areas) from where it is directed horizontally to the edge of the wound. The needle should penetrate the other edge of the wound at midstromal depth and exit the cornea 2 mm from the wound edge.

The interrupted sutures should be placed every 2 mm.

Scleral wounds should be closed similarly, using meticulous technique and midstromal depth placement of sutures. Noncolored sutures are usually used on the scleral wound, since these will remain buried after the conjunctiva is closed.

Despite impressive scientific progress, the most significant advances in the treatment of war zone trauma have been rapid helicopter evacuation and definitive care hospitals in the immediate war zone. Barely minutes may elapse from battlefield to operating room, where the ophthalmologist can undertake measures to salvage the injured eye and to restore vision, whenever possible. Finally, the frequent occurrence of combined ophthalmic, neurosurgical, and maxillofacial injuries makes optimum treatment truly a team effort.

CHAPTER XXV

Wounds and Injuries of the Ear

Injuries to the ear are common in combat. Such injuries may be confined to the external ear in the form of contusions, lacerations, or avulsions. Blast or other injuries to the middle or inner ear may cause deafness which may be permanent and is a primary consideration in all treatment. These wounds often are overlooked or relegated a low priority of treatment in a busy aid station or field hospital. However, such injuries, if not appropriately treated, may result in prolonged morbidity or permanent disability.

Patients who have sustained injuries to the ear which include impairment of hearing should be protected from additional acoustic trauma until maximum recovery is assured. Temporary or permanent reassignment of duty may be necessary.

THE EXTERNAL EAR

Trauma to the auricle is usually quite obvious and, unless treated promptly, may result in considerable cosmetic deformity. Among the more common injuries are lacerations, avulsions, contusions, or thermal injury.

In simple lacerations, the auricle should be debrided carefully with minimum excision of only the devitalized tissues. The laceration then should be closed in layers, being careful to realine cartilage with absorbable suture material and the skin and subcutaneous tissues with a fine, atraumatic suture. All cutaneous sutures should be removed in 3 to 5 days.

If the auricle is partially avulsed, careful surgical debridement and reapproximation should be accomplished as soon as possible. In those instances when a portion of the auricle is missing, approximation of the anterior and posterior layers of skin over the exposed cartilage should be accomplished. Fragments of the auricle which are still present should not be sutured out of their normal anatomic alinement. In instances of total avulsion, the cartilage should be debrided of all overlying tissue and buried subcutaneously in the abdominal wall (or other suitable area) so that it may be used for reconstruction at a later date.

When there is a hematoma of the auricle, the hemorrhage is usually subperichondral in origin. Such hematomas are evacuated surgically followed by a sterile pressure dressing. The dressing should be removed at least every 48 hours and the wound inspected for recurrence of the hematoma.

Thermal injury should be treated by careful cleansing and application of topical antibacterial agents such as mafenide (Sulfamylon) on fine mesh gauze. Asepsis is critical.

In all of the cited injuries, systemic coverage with broad-spectrum antibiotics, tetanus toxoid booster, and aseptic technique are essential.

Lacerations of the external auditory canal or fractures through its bony portion are less obvious and may be overlooked. Thus, they often do not become apparent until secondary infection has occurred. The external canal should be cleansed as aseptically as possible and a cotton or gauze wick impregnated with a broad-spectrum antibiotic inserted. Such patients should then be referred to the care of an otolaryngologist because of the strong possibility of stenosis as the canal heals. If a laceration of the external auditory meatus is recognized early, however, precise initial suture repair is indicated.

The problem of external otitis, especially in tropical and subtropical climates, is well known to combat physicians. As innocuous as this entity may seem, it has caused considerable morbidity. In such circumstances, the canal wall skin becomes macerated, affording an excellent culture medium for secondary infection. The organisms most commonly encountered are various species of *Pseudomonas* and *Proteus* with an occasional *Staphylococcus*. Thus, thorough cleansing plus broad-spectrum

antibiotics, both topically and at times systemically, is the treatment. Such infections are often extremely painful, requiring analgesics.

THE MIDDLE EAR

Injury of the tympanic membrane, which is common, is often associated with other, much more serious, injuries of the middle ear. The damage may be caused by direct penetration of a missile, by a fracture of the base of the skull involving the tympanic ring, or by sudden compression of the air in the external auditory meatus as the result of blast (see ch. V). A blast injury may cause a small hemorrhage in the substance of the membrane, rupture of the outer fibers, or a linear tear, or it may result in complete disintegration. The great risk is secondary infection, with deafness likely to be the end result.

Injury of the middle ear often is not attended with clear-cut symptoms. When it is suspected, the ear should be examined under aseptic precautions, with good illumination.

If rupture has occurred, instrumentation, drops, and syringing are all contraindicated. Wax is left in situ unless pain, deafness, or both require its removal; this is seldom necessary in a forward hospital.

Treatment in the forward area consists of simple protection of the ear with a sterile dressing or a loose packing of sterile cotton. If the pinna is also damaged, the meatus should be packed with sterile cotton while the outer ear is being cleansed.

Until the ruptured tympanic membrane has healed, every precaution is taken to avoid nasopharyngeal infection. The patient is warned not to blow his nose. If suppuration occurs, it must be vigorously treated by standard measures to prevent chronicity.

AERO-OTITIS

Aero-otitis is often encountered in flying personnel. Symptoms usually occur on descent when edema of the eustachian tube mucosa prevents equalization of pressure within the

middle ear. This can result in symptoms varying from mild pain and slight hearing loss to severe pain and extreme vertigo. When it occurs, topical and systemic nasal decongestants coupled with frequent Valsalva maneuvers often will reverse the process. On occasion, a myringotomy may be required. Prompt recognition and treatment will often sharply decrease the associated morbidity. The condition is more prone to occur in those with upper respiratory infections.

THE INNER EAR

Trauma to the inner ear may occur in combination with the above injuries or as an isolated injury secondary to blunt trauma. Such injury may be accompanied by total hearing loss, severe vertigo, high-pitched tinnitus, or facial nerve palsy. These injuries should be treated symptomatically and evacuated to the care of an otolaryngologist as soon as possible. If a basilar skull fracture is suspected, the use of antibiotics (usually penicillin) and decongestants is mandatory from first detection. In all of these instances, the patient should be cautioned against blowing his nose.

CHAPTER XXVI

Wounds and Injuries of the Neck

Wounds of the neck, because of the large number of vital structures within a compact area, are frequently complicated emergencies. Injuries to the neck can involve the larynx, trachea, pharynx, esophagus, major vessels, multiple nerves, cervical spine, and cord. Asphyxiation and severe hemorrhage are commonly associated. Most such patients require oxygen. They should be transported in a semisitting position unless contraindicated by other injuries. With any wounding agent, many systems can be injured at the same time. Lesions of the oropharynx or esophagus, communicating via the fascial planes of the neck with the mediastinum, may allow bacterial contamination and lead to mediastinitis. Foreign bodies carried into the soft tissues may cause further contamination. The hallmarks of good management of neck wounds are adequate incisions, generous exposure, and careful debridement followed by wide drainage. Small injuries to skin and fascia may be associated with more severe injuries to deeper structures. Neck wounds are often associated with oral, mediastinal, and pulmonary injuries. These injuries may initially be occult but will demand attention. Antibiotics are given to all patients with deep neck wounds.

ANESTHETIC CONSIDERATIONS

Preoperative medication should include atropine, to decrease secretions in the respiratory tract, and morphine, to reduce the laryngeal and cardiac reflexes, though morphine should be used with caution since it depresses the cough reflex. After the airway is controlled, general anesthesia is utilized according to techniques previously described.

WOUNDS OF THE LARYNX AND TRACHEA

Serious wounds of the larynx and trachea may present in the following ways:

1. Asphyxia. Asphyxia is an indication of laryngotracheal damage, with serious obstruction. The obstruction may be caused by destruction of the larynx, the fragments of which form obstructing flaps; by hemorrhage, which blocks the airway with blood or clots; or by laryngotracheal edema caused by trauma or infection. Restlessness may occur in these patients and is due to cerebral hypoxia either from airway obstruction or hemorrhage.

2. Dyspnea. Dyspnea may result from lesser damage to the larynx or trachea. The diagnosis of asphyxial injuries is immediately apparent, but injuries which cause dyspnea can often be found only by careful examination. The most common symptoms and signs, in addition to dyspnea, are dysphonia, laryngeal cough, hemoptysis, dysphagia, and excess mobility of the larynx. Roentgenologic examination of the laryngeal and tracheal cartilages, which are always ossified to some degree in adults, and preoperative laryngoscopy are of diagnostic assistance.

3. Subcutaneous emphysema of the face and neck. Retropharyngeal swelling, although frequently detected on physical examination, is demonstrable on biplanar soft-tissue X-ray films. Narrowing or distortion of the air column by a soft-tissue mass assists in this diagnosis.

All injuries of the trachea and larynx are serious. Diagnosis is confirmed by laryngoscopy or bronchoscopy, which should be performed at the slightest suspicion of injury. These examina-

tions often are done at the time of airway control, at which time an endotracheal tube may be inserted. The early use of this procedure precludes the performance of a hasty tracheostomy. Emergency tracheostomy, however, may be necessary in injuries that crush or distort the larynx or hypopharynx so that the airway cannot be visualized endoscopically. In such cases, urgent decompression of the deep subfascial space may also be necessary to relieve pressure on the airway. Many laryngeal injuries are ignored or undiagnosed once tracheostomy is performed, with subsequent serious loss of function, much of which may be prevented by early diagnosis and treatment.

Careful and conservative debridement of laryngotracheal injuries is emphasized. Following debridement, the fragmented larynx or trachea should be reapproximated and an intraluminal stent utilized to maintain the anatomical architecture. Late tracheal and laryngeal stenosis from injudicious and excessive removal of tissue, particularly cartilage and mucosa, must be prevented. Care must be taken to identify associated wounds of adjacent structures such as esophagus, pharynx, and major vessels.

Airway control with either tracheostomy or endotracheal tube requires constant aftercare to avoid sudden obstruction with asphyxia. Proper tube size is important. Too small a tube can result in gradual respiratory insufficiency, leading to hypoxia and cardiac arrest. Overinflation of tube balloons must be prevented to avoid damage to tracheal tissue.

WOUNDS OF THE PHARYNX AND ESOPHAGUS

The pharynx and esophagus are often involved in injuries of the neck, with the real possibility of contamination of the deep fascial planes of the neck and the mediastinum. Small lesions of the posterior pharynx and esophagus are often overlooked in the presence of other neck injuries and can lead to severe morbidity and death. Examination must be thorough and includes endoscopy. Any penetrating injury, however small, must be suspect. Soft-tissue X-ray films may be useful as previously

described. Radiopaque contrast media may demonstrate leaks not apparent by other means.

Management is based on surgical exploration, both to identify lesions and to debride and close lacerations of the mucosa and muscularis of the pharynx and esophagus. Double-layer closure of the defects is the treatment of choice, followed by adequate external drainage. Wide wounds of the pharynx or esophagus which cannot be closed will need either marsupialization or wide drainage. Nasogastric intubation is necessary, to decrease chances of contamination following regurgitation and, later, for feeding purposes.

VASCULAR INJURIES

Many injuries of the major neck and mediastinal vascular structures are fatal. Venous injuries have the added risk of air embolism. Serious vascular injuries may be masked in the patient with multiple injuries who is in severe shock. These may become apparent at any time after resuscitation has begun. Hemorrhage from neck wounds may be hidden as a hemothorax in combined injuries. Suspicion of injury to the vascular structures requires early exploration. Access to the great vessels in the chest is obtained by an anterior thoracotomy in the third interspace on the involved side permitting immediate tamponade. Definitive exposure of this region is then accomplished by a midline sternal splitting extension (fig. 25). This exposure also can be obtained by connecting the neck incision to a full midline sternotomy incision.

The following points in the care of vascular injuries are emphasized:

1. The mortality from uncontrolled hemorrhage is second only to asphyxiation in injuries to the neck. Airway control and hemostasis are, therefore, the initial steps.

2. Serious vascular injury often presents as a gradually enlarging hematoma, which can encroach upon the airway. This encroachment is produced by the expansion of the hematoma within the closed triple-layered deep fascial compartments of the neck. The fascial arrangement also prevents outward ex-

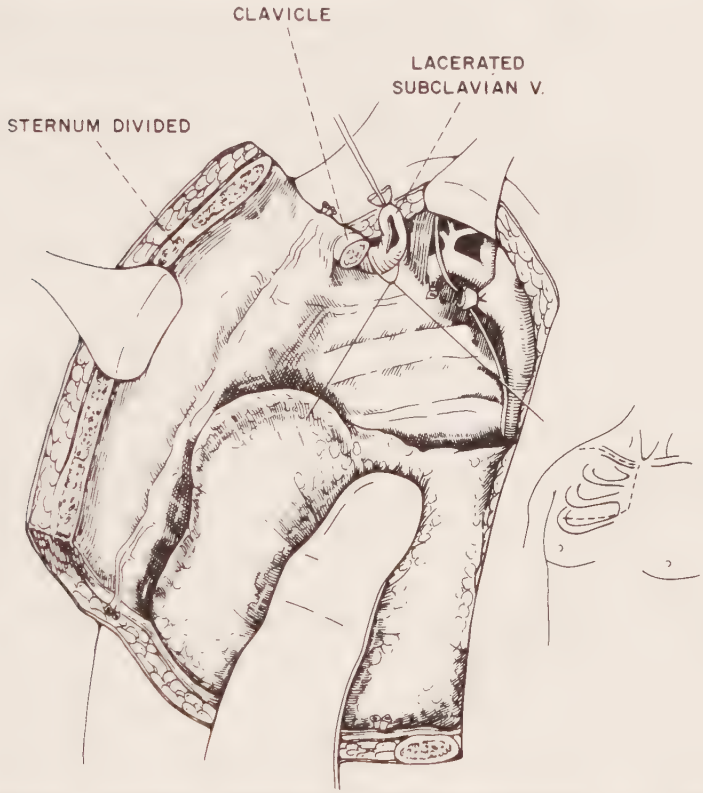


FIGURE 25.—Exposure of subclavian vessels by mobilization of a chest wall flap. The medial aspect of the clavicle is resected. The third interspace is joined to the incision by a median sternotomy.

pansion of extravasating blood, sometimes making the diagnosis of vascular injury difficult.

3. The penetrating wound of the neck, because it may involve vascular structures, requires definitive surgical exploration. Exploration should include the carotid and internal jugular systems. Adequate exposure with proximal and distal control is the important technical consideration in vascular repairs.

4. Lateral repair or end-to-end anastomosis after debridement of the injured wall of any artery is preferred. If this is impossible, autogenous vein grafts may be used to bridge an arterial defect. The use of an internal or external shunt to maintain

cerebral circulation during repair is preferred. The importance of adequate oxygenation and maintenance of blood volume cannot be overemphasized.

5. The external carotid system may be ligated without morbidity. Ligation of the internal carotid artery may be the safest procedure for patients with an injury to this vessel and an *already established neurologic deficit* (see ch. XVII).

6. Ligation of the internal jugular system is indicated when lateral repair is not possible.

NERVE INJURIES

Nerve injuries should be identified and noted for possible delayed repair. In general, immediate repair of traumatic nerve defects is not recommended. Even when ideally treated by delayed neurorrhaphy, the proximal location of the injury to the nerves in the neck precludes significant success. High-velocity missile wounds of the neck may cause spinal cord injury resulting in quadriplegia.

EMERGENCY TRACHEOSTOMY

Tracheostomy can be a lifesaving procedure and has proven its worth many times over (fig. 26). However, it requires a thorough knowledge of anatomy and must be performed many times before it can be done quickly and safely.

Adequate lighting for tracheostomy is essential. Positioning is also very important in tracheostomy. The patient lies supine, with the shoulders elevated by sandbags or folded towels, so that the neck is extended. Local anesthesia is usually utilized.

The incision may be longitudinal or transverse. The transverse incision will insure a better cosmetic result, but the longitudinal incision is almost bloodless and there is more rapid exposure of the trachea with it. It is made in the midline, through the skin and platysma, from the cricoid cartilage to the suprasternal notch. The strap muscles are separated in the midline by blunt dissection. When they are retracted, the

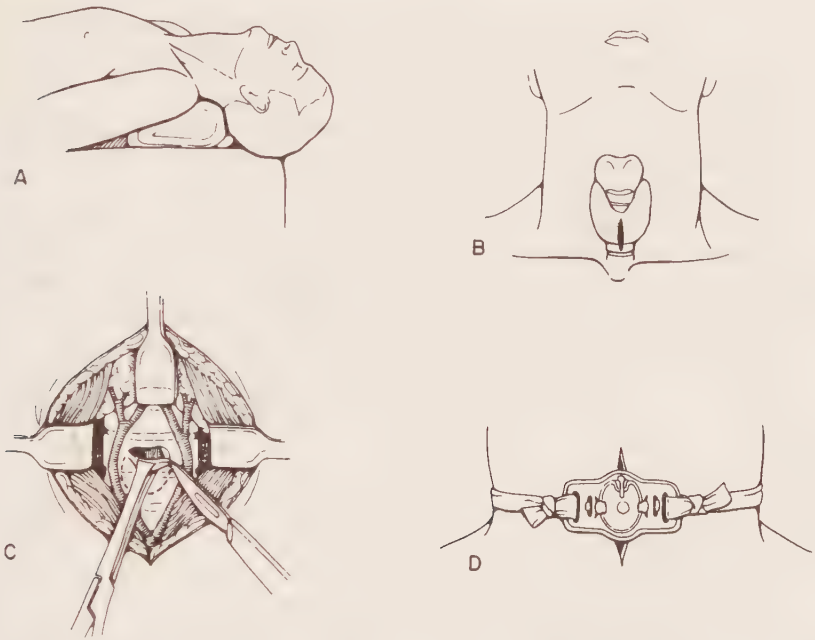


FIGURE 26.—Technique of tracheostomy. A. Position of patient with neck hyperextended. B. Location of incision. C. Thyroid isthmus elevated, pretracheal veins separated, window incised in trachea. D. Tracheostomy tube in place.

trachea is exposed. If the isthmus of the thyroid is encountered, it is displaced upward or downward.

If cocaine is available, a few drops of 4-percent solution are injected into the tracheal lumen to reduce the cough reflex. The pretracheal fascia is incised and stripped laterally as necessary to expose the underlying cartilages.

The only ideal level at which to cut the trachea is at the level of the second and third tracheal rings. The trachea is held firmly with a tenaculum while a small circular area of the tracheal cartilage is removed with a No. 11 knife blade. The stoma should be the precise size of the tube to be inserted. A heavy silk suture passed through one edge of the tracheal incision may be used later as a retractor and guide when early tube change is required. The adult male can accommodate a tracheostomy tube size between No. 5 and No. 8. Smaller tubes give

excessive airway resistance and can lead to hypoxia. After the tube is put into position, the skin incision is closed loosely with interrupted sutures.

Suction should be available at operation to remove secretions from the trachea. If it is not at hand, the head should be lowered as soon as the trachea has been opened.

In extreme airway emergencies, when the medical officer has had no experience with tracheostomy or endotracheal intubation or has no assistance, cricothyroidotomy must be performed. The thyroid cartilage is identified by finger palpation, and the cricoid cartilage, which is the smaller protuberance just below it, is similarly identified. The space between these cartilages, which is avascular, is incised transversely, and the tracheal lumen is readily entered through the incision. A rubber or plastic tube is inserted through the opening and sutured to the skin. If a tube is not available, a clamp can be used temporarily to keep the incision open.

Complications associated with tracheostomy are more frequent than realized and should be mentioned:

1. Subcutaneous emphysema with possible pneumomediastinum or pneumothorax.
2. Immediate and delayed severe hemorrhage from innominate, subclavian, and carotid vessels.
3. Tracheoesophageal fistula.
4. Tracheal and laryngeal stenosis.
5. Asphyxia from improper tubes.

CHAPTER XXVII

Wounds and Injuries of the Chest

Injuries of the chest, in addition to wounds of the thoracic wall, may include wounds of the lungs, trachea, major bronchi, esophagus, diaphragm, heart, and great vessels of the mediastinum.

These wounds may occur in any variety of combinations, including combinations of parietal and visceral injuries. In blast injuries or crushing injuries, there may be extensive visceral damage without any external wound of the chest wall. In most other chest injuries, there is also a parietal wound.

The prognosis is not entirely determined by the organs involved. A parietal wound, for instance, may be fatal in a crushing injury, and a cardiac wound may heal without any treatment. The only safe plan is to regard all chest wounds as potentially serious, however small the wound may be and however good the patient's condition may appear at the first examination. Any chest injury may be followed by disturbances of the pulmonary and cardiac functions which, if not promptly corrected, may be fatal.

PATHOPHYSIOLOGY

The pathologic process in all complications of thoracic injuries is primarily mechanical, which makes them generally easy to recognize and correct.

Pneumothorax

In most wounds of the lung, air escapes into the pleural space. Diminution in lung volume and vital capacity is proportional to the size of the pneumothorax. A small pneumothorax is usually absorbed within a few days and is of little consequence. A pneumothorax of considerable size usually can be diagnosed by reduced costal movement, hyper-resonance to percussion, and reduction of breath sounds on the affected side. The diagnosis is best made by chest roentgenogram.

Open pneumothorax.—Open pneumothorax occurs in wounds of the chest wall which permit air to enter and leave the pleural space during respiration (fig. 27). The term "sucking" aptly describes the sound made as the air passes through the wound.

In an open pneumothorax, atmospheric pressure causes collapse of the lung on the affected side. In addition, during inspiration, air is drawn from this lung into the opposite lung and the mediastinum is displaced toward the sound side. During expiration, air is blown from the lung on the sound side into the other lung and the mediastinum is displaced toward the wounded side. The quantity of air which reaches the lungs is, therefore, less than normal. The air contains an excess of carbon dioxide and a diminished proportion of oxygen. As dyspnea increases, the mediastinal movements become more violent, and venous return to the heart is impeded, so that cardiac output is diminished.

Tension pneumothorax.—In some instances, the pulmonary or chest wall wound, from which air escapes, is valvular, allowing air to enter the pleural space, where it is trapped. As a result, as the pneumothorax increases in size, the wounded lung is compressed and the mediastinum is displaced to the opposite side, with compression of the unwounded lung. Tension pneumothorax can be recognized by the usual signs of pneumothorax, combined with displacement of the trachea and the apex beat of the heart to the opposite side. Dyspnea is pronounced and cyanosis is evident. Subcutaneous emphysema is common. Patients with tension pneumothorax deteriorate rapidly and may die within minutes if relief of this condition is not obtained.

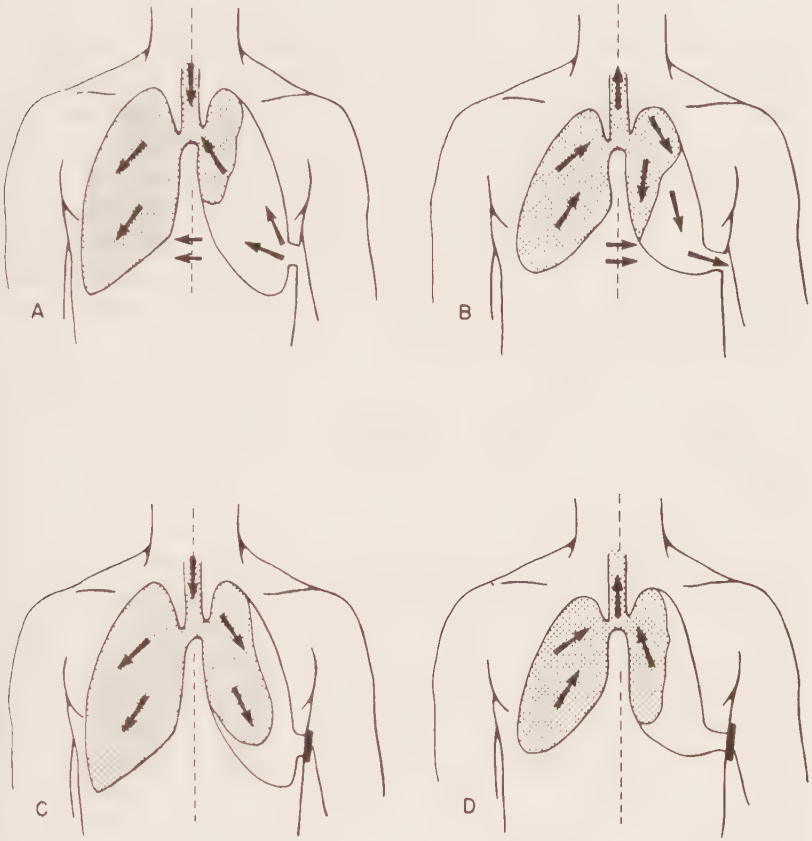


FIGURE 27.—Effect of open chest wound on respiration. A, B. Wound open. Note mediastinal shift and paradoxical respiration. C, D. Wound closure during inspiration and expiration, with disappearance of paradoxical phenomena.

Hemothorax

Blood may accumulate in the pleural space from wounds of the lung, mediastinal structures, and chest wall. Bleeding from the pulmonary parenchyma is usually slow and ceases spontaneously, though it is sometimes massive if hilar or systemic vessels are injured.

The volume of a hemothorax may be increased by effusion

into it from the pleura. As a result, although the hemothorax has the appearance of pure blood, it may contain only about half as much hemoglobin as is present in the circulating blood.

Clinical signs are those of a pleural effusion combined with loss of blood. The position of the mediastinum, which is ascertained by palpation of the trachea in the suprasternal notch and identification of the apical cardiac impulse, is an important indication of the size of the hemothorax. If a hemothorax develops rapidly enough to produce early dyspnea and mediastinal displacement, continued bleeding from a parietal or mediastinal vessel should be suspected.

The bulk of the hemothorax initially remains liquid, but a variable degree of clotting occurs later. Fibrin from the blood is deposited on the pleural surface and, if the hemothorax persists, may become organized and loculated, entrapping the lung.

Subcutaneous Emphysema

Air may escape from a pneumothorax into the soft tissues of the parietal wound. When the pneumothorax is of the tension variety, considerable air may be forced into the parietal tissues and crepitus will be readily apparent.

Subcutaneous emphysema, originating from a chest wound, is usually not dangerous in itself; the air is rapidly absorbed when leakage from the lung ceases. The patient's appearance is startling because of the inflation of the face, neck, trunk, and, sometimes, limbs. The worst features of the condition, clinically, are the inconvenience of the closed eyelids and the tenderness present over some loculi of air.

When subcutaneous emphysema begins at the suprasternal notch or is limited chiefly to the neck, it is likely that there is a wound of the trachea, a major bronchus, or the esophagus and that the escaped air has passed into the neck from the mediastinum.

Flail Chest

Effects similar to those of a sucking wound of the chest are produced without loss of continuity of the chest wall when an

area of the parietes loses its rigidity as a consequence of multiple fractures or double fractures of several ribs. Fractures of the sternum and of the costal cartilages lead to instability of the anterior chest wall, which tends to produce more paradoxic motion than is caused by posterolateral rib fractures where the chest wall is heavily protected by muscle layers and splinted by lying supine. When an unstable portion of the chest wall moves paradoxically with respiration, being drawn inward during inspiration and blown outward during expiration, the results are threefold: decreased pulmonary ventilation, mediastinal shift, and loss of the ability to cough and expel secretions.

Cardiac Tamponade

A wound of the heart may be followed by bleeding which fills and distends the pericardium. Increasing pressure of the pericardial hematoma obstructs the venous return to the heart and prevents normal filling of the atria.

Cardiac tamponade usually can be recognized by the weak pulse, the narrow pulse pressure, and the characteristic neck vein distention. If blood is not released from the pericardium as an urgent measure, death from inadequate cardiac output is inevitable.

PRINCIPLES OF MANAGEMENT

The treatment of chest wounds is based upon the general principles of management of all wounds, combined with the special principles related to the respiratory and circulatory disorders to which thoracic wounds may give rise. These principles are as follows:

1. Normal pleural and pericardial pressures must be maintained and the pleural space must be kept empty.
2. The bronchial tree must be kept clear of retained blood, foreign material, and bronchial secretions.
3. Minute ventilation sufficient for adequate oxygenation and removal of carbon dioxide must be assured. If oxygen ad-

ministration by nasal catheter is inadequate, more complex measures, including the use of tracheal intubation or tracheostomy and controlled positive pressure breathing, may be required.

4. The amount of hemorrhage must be estimated and blood replaced as necessary. After pleural drainage is established, hourly measurement of the blood loss from the chest is carefully recorded. This measurement is crucial in determining the need for emergency surgery to control hemorrhage.

MANAGEMENT IN THE DIVISION AREA

Surgical and radiographic facilities are not available in battalion aid stations. Therefore, treatment of chest wounds at this level must be limited to first aid and lifesaving measures. Such lifesaving measures include relief of respiratory obstruction, occlusive dressing to close an open pneumothorax, stabilization of a flail chest, relief of tension pneumothorax by the urgent use of a large bore needle or chest tube, and treatment of pericardial tamponade by pericardiocentesis.

Airway control.—Airway control may be difficult in a patient with injury to the upper airway, a deeply comatose patient, or a patient with severe flail chest. If laryngoscope and endotracheal tubes are available, endotracheal intubation is preferred initially. Both orotracheal and nasotracheal tubes may safely remain in place for 72 hours. Thus, tracheostomy can be performed as an elective procedure under good conditions at a later time. One of the greatest advantages of endotracheal intubation as a routine procedure is that it markedly reduces the number of tracheostomies required since airway problems in many patients may be resolved within 48 to 72 hours. An exception to the above is a patient with a severe flail chest, who almost always requires primary tracheostomy.

Closed tube thoracostomy.—If physical examination discloses signs of blood or air within the pleural space, intercostal tube drainage should be promptly established. Recent combat experience has proven that early and adequate evacuation of the pleural space by large chest tubes is essential. A large bore chest tube, e.g., 42 Fr, should be placed through the lateral

chest wall in the midaxillary line for removal of blood and fluid. A similar anterior tube, placed in the second interspace, should be used for removal of air from a significant pneumothorax. The tubes may be connected to a Heimlich chest drain valve and, from this valve, to a plastic bag or rubber glove. If a Heimlich valve is not available, a flutter valve may be fashioned from the finger of a rubber glove or a condom. As soon as possible, however, sterile underwater seal drainage and pleural suction of 20 to 30 cm of water should be applied. A chest roentgenogram should be taken as soon as possible to confirm proper location of the tube and adequate evacuation of the pleural space.

Flail chest.—As noted above, a severe flail chest requires immediate tracheal intubation or tracheostomy and will also require the institution of positive pressure breathing as soon as possible. To prevent the development of tension pneumothorax, a properly functioning chest tube should be in place before instituting positive ventilation. Lesser degrees of flail chest may be stabilized temporarily by strapping the injured segment with a firm dressing. This dressing should be over only the involved area of the chest wall so that it will not limit the function of the intact hemithorax.

Pericardial tamponade.—If there is any evidence of cardiac tamponade, the pericardium must be aspirated. The needle is inserted in the angle between the xiphisternum and the costal margin and is passed upward and backward, at an angle of 45 degrees into the pericardium. It is necessary to remove only sufficient fluid to improve the patient's blood pressure and pulse volume. The pericardial sac need not be completely evacuated. Repeated aspirations are often necessary until thoracotomy can be performed. Therefore, the patient should be evacuated as promptly as possible to the nearest surgical unit.

Thoracoabdominal wounds.—In 25 percent of thoracic wounds, there is abdominal involvement. The thoracic component of a thoracoabdominal injury requires the same treatment as any chest wound. One should keep in mind that the diaphragm has been lacerated and that herniation of abdominal contents into the chest may occur. The physical findings of traumatic diaphragmatic hernia can be confused with those of

tension pneumothorax or hemothorax and, therefore, a chest roentgenogram should be obtained as soon as possible. Viewing a chest film before placement of a chest tube is a wise precaution when it is possible to do so. These patients have a particularly high priority for evacuation because major surgery for the injured diaphragm and the associated abdominal component is mandatory.

Wound management.—Surface wounds are dressed in the usual manner. An open sucking wound must be closed immediately. This is best accomplished by an occlusive petrolatum-impregnated gauze dressing. A tension pneumothorax may occur after occlusion of a sucking chest wound, in which case a chest tube with closed drainage may be required.

Control of pain.—Chest injuries are often extremely painful. Voluntary splinting of the chest because of pain frequently leads to retained tracheobronchial secretions and inadequate minute ventilation. Intercostal nerve blocks with a local anesthetic agent are effective in restoring adequate cough and ventilation without depression of respiration, as is common with the use of narcotics. Narcotics may be used, however, in small doses intravenously to improve the patient's ability to ventilate and cough.

EVACUATION

After first aid measures have been completed, casualties with chest wounds are evacuated as promptly as possible. Maximum priority is given to those with continued intrapleural bleeding, cardiac tamponade, or thoracoabdominal injuries. Management of the airway, oxygen therapy, and proper handling of chest tubes must be provided for during transportation. To avoid tension pneumothorax or clotting in the tube, chest tubes should not be clamped while the patient is transported. Instead, all tubes should have one-way valves attached as described above. Whenever possible, patients with chest injuries should be transported with the head and chest elevated 45 degrees; this position significantly improves pulmonary ventilation and the patient's ability to cough.

MANAGEMENT IN THE FORWARD HOSPITAL

Treatment initiated in the division area is continued when a hospital with surgical facilities is reached. If not done previously, roentgenograms are now made to demonstrate the character and extent of the damage, to show the position of metallic foreign bodies, and to assist in planning the exact operative procedure required. Necessary supportive measures are initiated or continued.

Most chest injury cases ideally are managed without thoracotomy. The mainstay of this treatment is closed tube thoracostomy, which is employed for a period necessary to obtain inflation of the lung and clearance of the pleural space. The tube may be removed when there is no longer evidence of air leak and when blood and fluid are no longer present. This is the best means of avoiding pleural infection.

Open thoracotomy is not performed routinely in wounds of the chest. It is limited to one or more of the following indications:

1. Continued intrathoracic bleeding.
2. Massive continuing air leak.
3. Mediastinal injury (great vessels, trachea, or esophagus).
4. Debridement and closure of large chest wall wounds.
5. Cardiac wounds that are immediately life-threatening or that have recurring cardiac tamponade after aspiration. Recent combat experience indicates a high percentage of favorable results when operation is performed early in these cases rather than employing repeated pericardiocentesis.
6. Use of thoracotomy for closure of the right diaphragm when not possible at celiotomy.

Continued or massive intrathoracic bleeding has been the major indication for thoracotomy in nearly all experience. The next most frequent indication for thoracotomy is a large defect of the chest wall which requires surgical attention. Air leak and mediastinal injuries are uncommon indications for thoracotomy.

Anesthesia.—All thoracotomies are done under general anes-

thesia, with controlled positive pressure respiration via an endotracheal tube, or via a previously established tracheostomy.

Technique.—Wounds of the heart and proximal great vessels are best approached through a median sternotomy; other wounds are best approached through a standard posterolateral thoracotomy incision. Adequate exploration and evacuation of the pleural space can be performed through an existing large wound of the chest wall. Chest wall defects should be closed with adjacent normal tissue when possible, devising muscle flaps to close the defect without tension. If airtight closure has been obtained with underlying tissue, the skin and subcutaneous tissue should be left open for delayed primary closure. Rib fragments should be removed and injured rib ends should be smoothed to prevent subsequent lung damage. Following thoracotomy, intercostal tube drainage is instituted with underwater suction.

A formal posterolateral thoracotomy is used for those cases requiring exploration. If intrapleural bleeding has persisted, blood and blood clots, which are frequently present in large amounts, are removed from the pleural space, and the bleeding vessel, which is usually an intercostal artery or the internal mammary artery, is secured. Bleeding from the lung usually ends when the lung is inflated and comes into contact with the chest wall, but in lacerated wounds, ligation of bleeding vessels may be necessary.

Foreign bodies in the pleura are removed, as are any others which are readily accessible. No search should be made for inaccessible foreign bodies which may have been demonstrated by X-ray films.

Pulmonary resection is seldom necessary. A severely contused lung has remarkable powers of recovery. Any single great vessel in the pulmonary hilum may be safely ligated without risk of pulmonary necrosis. If, however, a segmental or lobar bronchus has been damaged, resection of the lobe supplied by the bronchus may be necessary. Otherwise, leaks are closed by ligation of small bronchi and by suture of main bronchi.

If thoracotomy has been done on the indication of recurrent cardiac tamponade, the pericardium is opened from apex to base. Although the wound in the heart is always small—other-

wise the patient would not have survived—a forceful stream of blood may shoot out through it when the pericardial pressure is released. Hemorrhage is controlled by digital pressure while sutures are inserted to close the wound; the finger is slowly withdrawn as sutures are tied. The pericardium is closed loosely so that fluid can escape freely from it into the pleural space, whence it can be aspirated.

Esophageal perforations are closed by suture in two layers if possible. Because of a high incidence of fistula formation despite repair, gastrostomy or jejunostomy should be considered and the chest tube should remain in place for approximately 7 days. Oral feeding may be instituted following a negative esophagogram in approximately 1 week.

Closure.—The wound is closed in layers by the usual technique. Soft tissues covering large defects are stabilized by firm dressings and strapping, as in stove-in chest.

Thoracoabdominal wounds.—The diagnosis of thoracoabdominal wounds may be difficult in that thoracic wounds alone may produce abdominal rigidity and pain referred to the upper abdomen. The course of the missile and the location of foreign bodies as demonstrated by roentgenogram, keeping in mind how high the dome of the diaphragm rises into the chest during expiration (level of the nipples), will help to identify the combined wound. Another important anatomic feature is the depth of the pleural cavity in the costophrenic sulcus, posteriorly and laterally. Therefore, upper abdominal viscera are commonly injured in wounds of the chest. The majority of thoracoabdominal wounds can be managed by intercostal tube drainage of the pleural space and celiotomy to treat the intra-abdominal injuries and suture the diaphragm from below.

If the thoracic component of the wound is large, it will require debridement and closure with adequate surgical evacuation of the pleural space. In the course of this procedure, one should resist the temptation to perform extensive abdominal exploration through the diaphragm. It is preferable to close the chest incision and make a separate celiotomy incision which will allow more adequate exposure for thorough diagnosis and treatment of all intra-abdominal injuries. Occasionally, a small separate thoracotomy incision is required to repair the right diaphragm when this is not possible from below.

POSTOPERATIVE MANAGEMENT

Great attention must be placed in the postoperative period to the maintenance of adequate pulmonary ventilation and the removal of tracheobronchial secretions by coughing and suctioning. Oxygen therapy should be administered at least during the first 24 hours after operation. Nasotracheal suctioning and, possibly, bronchoscopic aspiration may be necessary for the removal of secretions or relief of bronchial obstruction. Patients who cannot ventilate adequately on their own will require the assistance of a volume-cycled respirator. The administration of blood and other intravenous fluids should be cautious during the postoperative period because of the frequency of the "wet lung" syndrome which is aggravated by overadministration of fluid. If hypoxemia develops or the postoperative chest roentgenograms show any evidence of pulmonary edema, potent diuretic agents should be given and fluids should be rigidly restricted. Patients with the "wet lung" syndrome should be transferred promptly to an intensive care unit equipped for intensive respiratory management (see ch. XXVIII). Continuation of oxygen therapy and positive pressure respiration is essential for the safe transport of such patients.

Chest roentgenograms should be taken repeatedly to assure proper location of pleural tubes and expansion of the lungs. Whenever possible, the patient should not be evacuated until 72 hours after removal of chest tubes, and a chest film should be taken and interpreted immediately before evacuation.

CHAPTER XXVIII

Wounds and Injuries of the Abdomen

The casualty with an abdominal wound must be evacuated promptly to an installation with surgical capability. However, certain lifesaving procedures must be accomplished in the division area before the patient is transported (see ch. XIV).

Without adequate and prompt treatment, intra-abdominal injuries are almost always lethal. Hemorrhage from wounds of major vessels within the peritoneal cavity is rapidly fatal without surgery. Wounds of the gastrointestinal tract, liver, pancreas, kidney, and bladder result in chemical or bacterial peritonitis unless immediate corrective surgery is performed.

With rare exceptions, the treatment of abdominal wounds is surgical. The eventual outcome of any patient with an abdominal wound is most closely correlated with the number of intra-abdominal organs injured. For a given magnitude of injury, mortality increases with increasing time from injury to operation. Although the tactical and logistical situations must always be considered, for optimum results, laparotomy should be performed as soon as medically feasible.

Patients with wounds of the abdomen tolerate poorly the added trauma of movement and evacuation. An individual who has had emergency surgery because of intra-abdominal injuries, therefore, should remain for several days in the facility where the operation was performed. Before evacuation, bowel function should be reestablished and nasogastric suction should no longer be required. If the patient cannot be retained be-

cause of the tactical situation, he should be moved to the closest secure installation where essential postoperative care can be continued in an orderly manner.

PREOPERATIVE SORTING

Examination

When casualties with abdominal injuries arrive at a hospital with surgical capability, the first procedure is careful assessment of the patient's general condition and local wounds. Such wounds may occur in the perineum, buttocks, and back, areas which are frequently overlooked in the evaluation of patients with abdominal wounds. If possible, the physical examination is performed without moving the patient from his litter. The findings on clinical examination at this time should be compared with the data recorded on the field medical card.

Blood for typing and crossmatching should be drawn. When time and facilities permit, other standard laboratory tests, such as urinalysis, hematocrit, and blood counts, may be obtained. However, emphasis should be directed primarily at resuscitation rather than at obtaining laboratory data. Roentgenologic examination to detect the position of foreign bodies, the trajectory of missile tracks, and unsuspected injuries may be helpful and timesaving in the operating room. A simultaneous intravenous pyelogram may be of value in determining injuries to the genitourinary system and the presence of renal or ureteral anomalies.

Priorities

Massive intra-abdominal hemorrhage has the highest priority for surgical treatment. For such patients, surgical treatment may be required as an integral part of resuscitation. Patients with lesser degrees of intra-abdominal hemorrhage and injuries of hollow viscera may be prepared for operation with preoperative fluids and blood appropriate to treat shock, and preoperative intravenous antibiotics. Operation then may be performed

on a deferred basis, keeping in mind the desirability of repairing the defects as early as possible to prevent the deleterious effects of continuing shock and peritoneal infection. When caseloads are high, such patients may be delayed for a short period of time; for example, 1 to 2 hours.

Cases of abdominal injury recognized late, either because of delay in diagnosis or evacuation, usually require operation, but have a lower priority. Patients whose condition is so poor that surgery offers no reasonable prospects of success have the lowest priority, but, if time permits, surgery may be undertaken on a selective basis.

OPERATION

Anesthesia

Adequate relaxation is essential for laparotomy to allow adequate exploration with the least degree of additional trauma. A high degree of expertise is mandatory to achieve this end. Endotracheal anesthesia is preferred to prevent aspiration and to assure an adequate airway. The technique used to administer the anesthetic should be that with which the anesthetist is most familiar (see ch. XV).

Associated Wounds

Unless grave internal hemorrhage is suspected, major wounds of the back or buttocks should be debrided before the abdomen is entered. To turn the patient into the prone position after laparotomy to perform such debridement may result in severe hypotension or cardiac arrest. If the back or buttock wounds were not cared for before laparotomy, an attempt to turn the patient to the lateral decubitus position should be carefully monitored. If this position is well tolerated, debridement of these wounds is accomplished; if not tolerated, the wounds are dressed and operation is postponed until the patient's condition stabilizes.

Associated minor wounds are not ordinarily treated until after abdominal operation is concluded, and they are cared for only if the patient's condition is so stable that he will not be harmed by prolongation of the anesthesia and the extra time required for the additional procedures. Failure to observe this precaution may prejudice his chance for survival.* Small, apparently superficial wounds of the abdomen and lower chest do not fall into this category. In these cases, the abdomen must be adequately explored to be certain that the peritoneal cavity has not been entered. Probing of the wound is not sufficient. These wounds must be debrided in the usual manner and left open for delayed primary closure.

Exploration and Control of Hemorrhage

A generous midline incision provides the quickest and best access to the entire abdomen. After the abdomen has been opened, a rapid but careful exploration should be carried out. It is most important that the investigation be conducted in an orderly systematic fashion; there is no other way of insuring that injuries will not be overlooked. If hemorrhage is occurring, its source is sought systematically by inspection and palpation of the liver, spleen, kidneys, stomach, mesentery, intestine, and other intra-abdominal structures. If necessary, the search is facilitated by delivering the entire intestine onto the abdominal wall. This maneuver is shocking, and expert anesthesia is required to make it safe. However, it may be extremely useful. It permits detailed inspection of the entire bowel. It provides better access to the liver and spleen. Finally, if spillage is occurring from small bowel perforations, exteriorization permits its exit externally, where it will do no harm, and prevents further contamination of the peritoneal cavity. If the wound involves the upper abdomen, an opening into the lesser omental sac should be made through the gastrocolic omentum, and the pancreas and posterior stomach wall should be examined.

Injuries of large intra-abdominal vessels are usually lethal. If the casualty survives to undergo an operation, repair or ligation is carried out as indicated. Management of vascular in-

juries, moreover, provides the opportunity to determine whether circulatory damage to the bowel has occurred. If the ligature of a mesenteric vessel is followed by circulatory inadequacy in the corresponding loop of bowel, resection is necessary.

After hemorrhage is controlled, including that from solid viscera, the intestinal tract is examined systematically from end to end. Particular attention is paid to the posterior surface of the stomach and the duodenum, as well as to the retroperitoneal colon. These are areas in which injuries easily can be overlooked. On the least indication, they must be inspected after mobilization of the fixed portions of the colon and retroperitoneal duodenum. Injury to the kidneys, ureters, bladder, and diaphragm also should be ruled out. Perforations which are found are temporarily occluded by intestinal clamps until the definitive procedures can be accomplished.

Wounds of Solid Viscera

Liver.—The principles involved in handling liver trauma include adequate drainage, suture for hemostasis, resection of devitalized tissue, and drainage of the biliary tree. All liver wounds should be afforded adequate dependent drainage. It is mandatory that the drainage incision be large and placed as far posteriorly as possible. The use of sump drains is recommended in hepatic trauma of any significant magnitude. Suture of liver substance is primarily to secure hemostasis and to stop biliary leakage. Sutures should be placed deeply into liver parenchyma to eliminate dead space. Care should be taken not to suture the liver capsule over an intrahepatic cavity which could provide a setting for future hepatic abscess or hemobilia. All devitalized liver tissue must be debrided and, in more severe cases, resective debridement may approach the point of total hepatic lobectomy.

For the purpose of outlining treatment, liver wounds may be arbitrarily classified into three types according to their severity. The first type is a wound created by low-velocity fragments penetrating the substance of the liver. Such wounds usually present with minimal to moderate bleeding and limited anatomic disruption of the liver. They are treated by limited

debridement, hemostasis, and external drainage. Only external drainage may be required.

The second type is one which is due to missile fragments causing shattering of the liver parenchyma and hemorrhage of a moderate to severe degree. Resective debridement, hemostasis, and effective external drainage are required. Biliary tract decompression may or may not be indicated. In this type of wound, surgical judgment is needed to decide if and how much resection of liver tissue will be necessary to control the hemorrhage.

The third type of liver wound is that caused by high-velocity missiles with extensive shattering or maceration of liver substance. This is always associated with severe hemorrhage, which should be suspected as coming from the hepatic veins or intrahepatic vena cava. There should be no hesitation to extend the incision into the chest for control of hemorrhage and prevention of air emboli. It is apparent when the wound is first evaluated that partial hepatectomy or lobectomy will be required. Any means of controlling hemorrhage which seems feasible should be used. These may include use of partial occluding clamps on the vena cava; external tape occlusion of the vena cava proximal and distal to the hepatic veins; occlusion by clamps upon the abdominal aorta, portal vein, and vena cava above and below the liver (fig. 28A); or the use of occlusion ballon catheters within the vena cava connected by an internal shunt (figs. 28B and C). This type of injury taxes the skill of the entire surgical team. Mortality is consistently high in this group (65 to 85 percent).

T-tube drainage of the common duct should not be used routinely but is acceptable in severe cases of hepatic trauma. This may help to decompress the biliary tree and, perhaps of greater value, offers an excellent means for later diagnostic study. T-tube decompression is not a substitute for adequate external drainage. If for some reason the common duct cannot be utilized, cholecystostomy may be considered. Neither T-tubes nor catheters should be removed before evacuation. Bleeding diathesis frequently occurs with serious liver injuries and may require fresh whole blood.

The following guidelines are presented:

1. The midline abdominal incision usually provides ade-

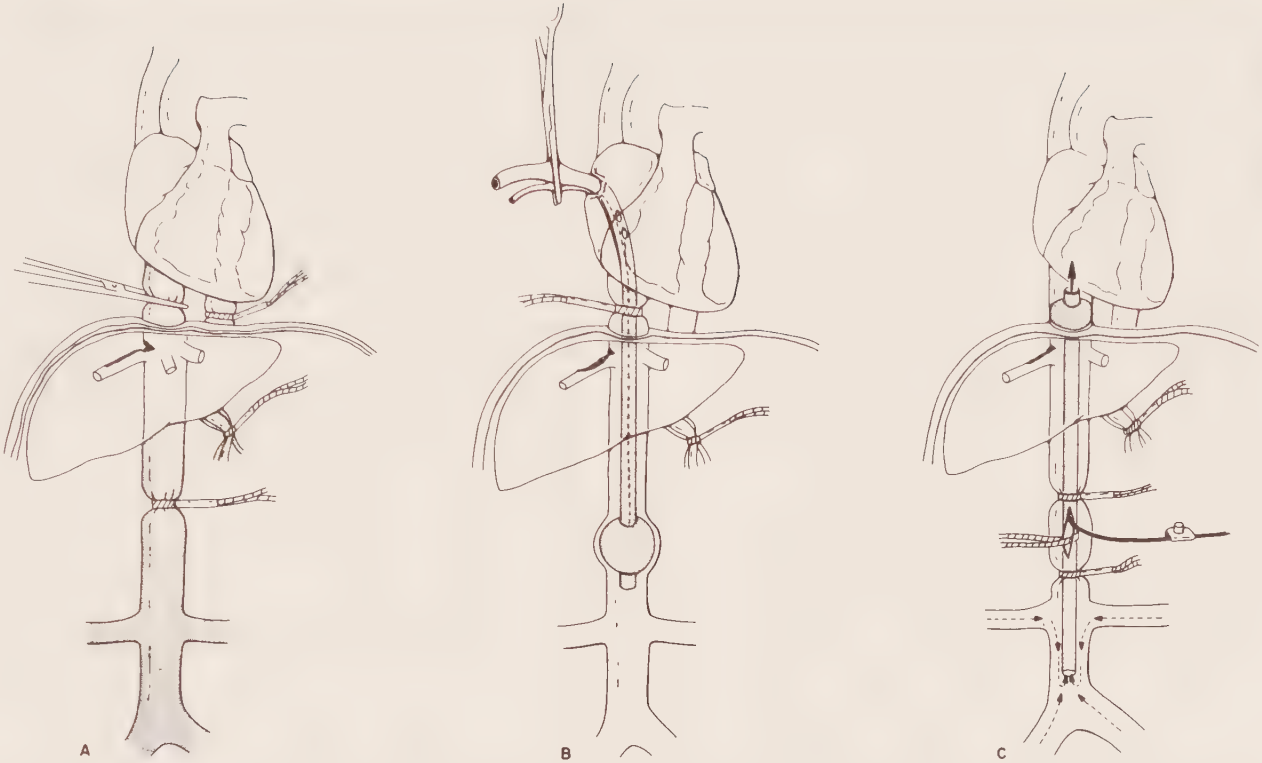


FIGURE 28.—Control of bleeding from caval and hepatic vein injuries associated with liver injuries. A. Four-clamp technique. B. Balloon tamponade via right atrium. C. Intracaval balloon catheter via suprarenal vena cava.

quate exposure. Thoracic extension may be necessary in treating more severe wounds.

2. Control of hemorrhage during resection can be facilitated by the use of hepatic inflow occlusion, which can be safely utilized for 15 minutes in the normothermic patient or up to 1 hour in the hypothermic patient (approximately 30° C).

3. The line of liver resection should be at the edge of devitalized tissue.

4. The use of packing to control liver hemorrhage is not recommended except in extreme circumstances.

5. Injuries to the dome of the liver frequently involve the diaphragm, which should be repaired to prevent biliary-pleural fistula. Repair of the diaphragm may require the otherwise infrequently indicated separate thoracic incision.

Major complications of liver wounds include secondary hemorrhage; infection, either subphrenic, subhepatic, or intrahepatic; and biliary fistula. These complications usually result from:

1. Improperly placed drains.

2. Inadequate drainage and failure to make an adequate drainage incision.

3. Failure to debride adequately the necrotic or devitalized liver tissue.

4. Strangulation of hepatic tissue by suture material.

5. Gross contamination of the hepatic tissues from associated colon, stomach, small bowel, and duodenal injuries.

Spleen.—Injuries to the spleen are uniformly treated by splenectomy. Subphrenic infection is the most common complication following splenectomy and usually is related to concomitant injuries to other organs such as the colon, pancreas, kidneys, or stomach. When neighboring organs are injured or there is any question regarding adequate hemostasis, the splenic fossa should be drained.

Pancreas.—Wounds of the pancreas are infrequent. A univisceral wound to this organ seldom occurs. In wartime, recorded frequency of injury to the pancreas is low because these injuries are usually lethal, due to the proximity of major vessels to the pancreas. Whenever there is a penetrating wound of the upper abdomen, the pancreas should be examined. When in-

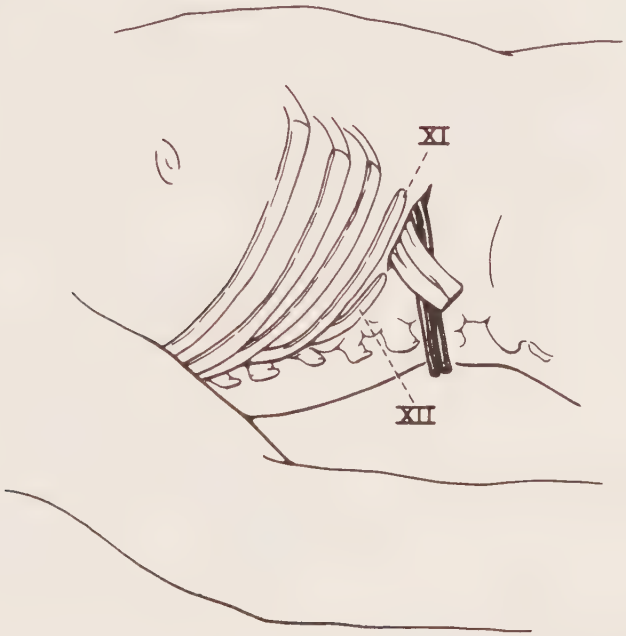


FIGURE 29.—For adequate drainage of the abdomen, drains must be placed in the most dependent portion of the peritoneal cavity. This is best accomplished through the posterior flank utilizing a combination of Penrose and sump drains.

jury is suspected, the gastrocolic omentum should be divided and the entire pancreas examined.

Minor injuries to the pancreas in which there is minimal contusion or laceration, without disruption of major pancreatic ducts, should be treated by generous dependent drainage through the posterior flank (fig. 29).

Severe injuries involving the neck, body, or tail of the pancreas in which there is maceration or laceration of portions of the pancreas with disruption of pancreatic tissue and ducts should be treated by resection of the distal pancreatic remnant with ligation of the proximal pancreatic duct and closure of the pancreatic capsule if possible. Posterior dependent drainage using sump drains is mandatory. Attempts at Roux-en-Y drainage of pancreatic remnants or efforts to preserve portions

of the tail and body of the pancreas ordinarily should not be attempted in war casualties. It is safer to resect the injured portion and establish good drainage.

Pancreaticoduodenectomy may be required in those casualties in which the duodenum and head of the pancreas are so severely injured that no other procedure is feasible. In any pancreatic injury, drainage is essential.

Wounds of Hollow Viscera

Biliary tract.—Wounds of the gallbladder and cystic duct are managed by cholecystectomy. Wounds of the hepatic or common bile ducts are managed by T-tube drainage. Some wounds lend themselves to immediate repair; but when large segments are missing, a Roux-en-Y choledochojejunostomy may become necessary to restore functional integrity. Trauma to the bile ducts is commonly followed by stricture. Reconstructive surgery in such complicated cases is performed later at a more suitable time and under optimal conditions.

Stomach.—Injuries to the stomach should be treated by adequate debridement and a two-layer closure. Occasionally, the stomach will be so severely injured that a partial gastrectomy will be required. In all casualties who have a penetrating injury to the anterior wall of the stomach, a second wound must be searched for by thorough examination of the posterior wall.

Duodenum.—Because of the high associated mortality, wounds of the duodenum are infrequently encountered by the surgeon. Injury to the duodenum is seldom univisceral but is associated with significant injury to the pancreas, vena cava, liver, stomach, or kidney. Because of the proximity to the major vessels of the abdomen, patients with wounds of the duodenum often have severe hemorrhage.

Minor wounds of the duodenum should be treated by debridement and simple closure after the posterior wall has been mobilized and examined. More severe wounds may be treated by partial resection and primary anastomosis, except the second portion of the duodenum which contains the ampulla of Vater. Pancreaticoduodenectomy should be used for those severe injuries in which the duodenum and the head of the pancreas are

so severely injured that no other form of therapy is feasible. In some cases where a defect in the duodenum precludes primary closure without tension, serosal patching with a loop of jejunum is an acceptable procedure. If this method is used, care must be taken to place the anchoring sutures far enough away from the edge of the defect in the duodenum to prevent necrosis and leakage. After repair of the duodenum has been completed, a retrograde catheter placed through the proximal jejunum should be passed proximal to the duodenal anastomosis to accomplish decompression. If loss of the distal duodenum is extensive, anastomosis with the jejunum can be established. Adequate posterior dependent drainage with sump tubes and Penrose drains is necessary.

Small intestine.—The small intestine is the most frequently injured intra-abdominal organ in combat. Wounds to the small intestine are often multiple and tend to occur in pairs; however, tangential wounds are possible.

Most small bowel perforations can be treated by a single-layer transverse closure. When an injury is more severe than simple perforation, resection may be necessary. Resection is preferable to repair in the following circumstances:

1. When perforations are so close together that repair would jeopardize the regional blood supply.
2. When there are multiple injuries in a short segment and individual repair would take an undue amount of time.
3. When the bowel has been torn from its mesenteric border or the regional blood supply has been impaired by injury to the mesentery.

End-to-end anastomosis is the preferred technique. A single layer of invaginating sutures is rapid and adequate (fig. 30). Thorough evaluation of mesenteric blood supply is mandatory before closure of the abdomen. Single arcade vessel injuries may be ligated. However, multiple vessel damage and large areas of hematoma must be evaluated carefully, and if there is a deficient blood supply to a portion of the bowel, resection and anastomosis at viable levels are mandatory.

Colon.—Wounds of the colon occur almost as frequently as those of the small intestine. Because of the more potent bacterial contamination which occurs with wounds of the colon and because of its poorer healing properties, the complication

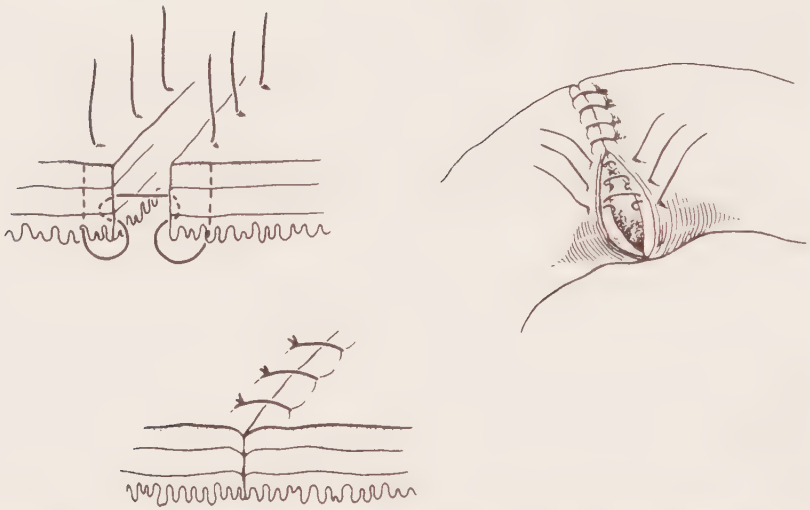


FIGURE 30.—An inverting single-layer suture technique is satisfactory for repair of small bowel injuries and anastomosis of small bowel after resection.

rate and mortality are higher. In the majority of cases, exteriorization of colon wounds is preferred (fig. 31). Not only are gross disruptions exteriorized, but also contusions of the colon, which have a tendency to delayed breakdown. The major exceptions to this rule are injuries to the right colon and rectum. The loop of exteriorized colon should never be closed at the time of the initial surgical procedure. Colostomies or exteriorized colon bearing an open wound should be opened adequately as soon as practicable to afford decompression. This can be done safely in approximately 48 hours.

Wounds of the right colon should be managed in one of two different ways depending upon the extent of injury and the number of associated injuries:

1. Resection with ileostomy and mucous fistula. When there are severe injury to the right colon and associated injuries to contiguous organs, resection of the involved colon should be accomplished, followed by construction of a surgically matured ileostomy and end mucous fistula of the colon (fig. 32A, B). This is the preferred treatment for wounds of the right colon. The mesentery of the distal ileum should be sutured to the right

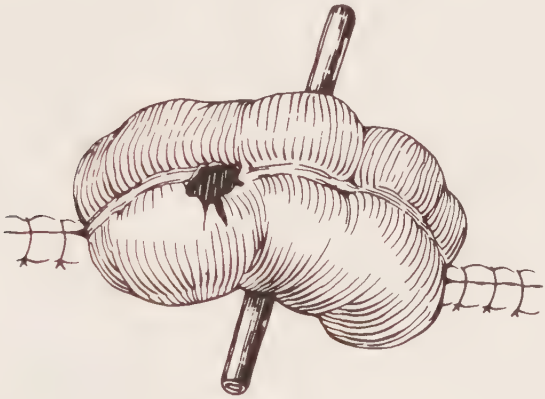


FIGURE 31.—In the majority of cases, exteriorization of colon wounds is preferable. A loop of colon is exteriorized over a glass rod or a rubber catheter.

peritoneal reflection and abdominal wall, when feasible, to preclude the development of an internal hernia, volvulus, or prolapse.

2. Resection and ileocolostomy. The involved segment of injured right colon should be resected and continuity reestablished by ileocolostomy in certain highly selective cases. The anastomosis is accomplished by a standard two-layer technique. This procedure should be reserved for injuries of the right colon in which there are no serious associated injuries to the liver, kidney, ureter, or duodenum. When this procedure is performed in the presence of severe multivisceral injury, particularly with liver involvement, a significant number of anastomotic leaks results in intra-abdominal abscesses and increased mortality. Because of the gross contamination which occurs in these severe wounds, establishment of adequate posterior dependent drainage of the right peritoneal reflection (gutter) and subhepatic spaces should be established. Drains should not contact the anastomosis.

Simple closure or placement of a cecostomy tube through a debrided cecal wound is rarely indicated in treatment of right colon wounds and may be considered only for the smallest perforations with no evidence of surrounding bowel contusion.

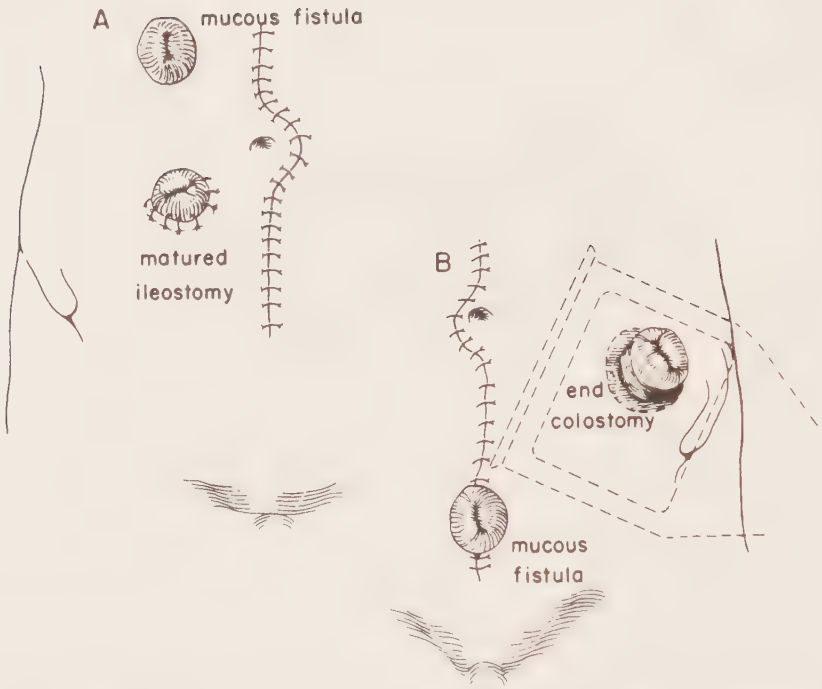


FIGURE 32.—A. A matured ileostomy, which will accommodate an ileostomy appliance, and a distal mucous fistula are indicated when there are severe injury to the right colon and severe associated injury to contiguous organs. B. The construction of an end colostomy and a distal mucous fistula which allows placement of an appliance over the proximal stoma is mandatory for diversion of the fecal stream in management of low sigmoid and rectal injuries.

These procedures must be accompanied by inspection of the posterior aspect of the involved colon for a second wound. Wounds of the right colon are not satisfactorily managed by exteriorization. The liquid and enzymatic nature of the effluent from such a bowel loop and the inability to maintain a secure skin-fitting appliance cause intractable problems with the abdominal wall and severe problems in fluid balance. This technique is mentioned only to condemn it. In general, this procedure should be avoided in favor of resection because of the mechanical difficulty encountered.

The transverse colon lends itself to exteriorization after in-

jury. Wounds to this portion of the colon can be treated in this manner. When severe disruptive injury has occurred or significant segments have been devitalized, resection and double-barreled colostomy should be performed.

Penetrating injuries to the left colon, like those of the transverse colon, usually can be exteriorized; however, in the fixed portions, surgical mobilization is imperative so that the exteriorized bowel is under no tension. This is easily accomplished in most instances. When severe injury has resulted in disruption or a devitalized segment, resection with end colostomy and distal mucous fistula should be accomplished. Small wounds of the distal left colon which cannot be exteriorized can be closed in two layers and a proximal diverting colostomy performed. When the left colon is extensively injured so close to the peritoneal reflection that exteriorization of the distal segment is not feasible, the injured colon is resected, a proximal colostomy formed, and the distal colon closed in two layers as a blind pouch. As on the right side, injury to retroperitoneal organs is also sought. Likewise, when there is associated contiguous organ injury, the left gutter and pelvis should be drained with multiple soft rubber drains.

When multiple wounds of the colon occur, the distal wounds are debrided and closed, or resected and anastomosed, and the proximal injured colon is formed into a diverting colostomy which allows the application of an appliance over the proximal stoma. Alternatively, when multiple wounds of the colon are relatively close together, a single resection with establishment of proximal end colostomy and a distal mucous fistula is performed. If the distal pelvic colon cannot be exteriorized readily, it should be closed leaving it within the pelvic cavity.

The wounded colon or the colostomy should be brought out through a separate incision, not through the traumatic wound or the surgical incision. The site of exteriorization or colostomy is placed to avoid further contamination of these wounds.

Rectum.—Wounds of the rectum have a high mortality and morbidity and are among the most disabling injuries encountered. Control of hemorrhage is often a major problem. Injuries to this portion of the colon are frequently associated with fractures of the pelvis, small bowel perforations, major nerve injuries, bladder perforations, and transections of urethra.

Failure to recognize and divert urinary tract injuries or debride devitalized bone fragments accounts for many complications. Although the rectal wound itself is usually well controlled, associated chronic pelvic osteomyelitis or urethral stricture may lead to long term pain and disability.

Adequate drainage by one of several methods must be accomplished at the time of the initial injury. When there is an injury to the lower sigmoid or rectum resulting in pelvic contamination, drainage of the cul-de-sac and pararectal area is facilitated by development of the pararectal and presacral spaces at the time of laparotomy. Adequate Penrose drains, and sump drains if desired, are subsequently placed through a vertical posterior Kraske incision (fig. 33). Free presacral drainage is enhanced by coccygectomy. The major error in treatment of rectal wounds is inadequate drainage through inadequate drainage incisions!

Rectal wounds demand an end colostomy and a distal mucous fistula with enough space between the two limbs of the colostomy to completely divert the fecal stream (fig. 32B). The

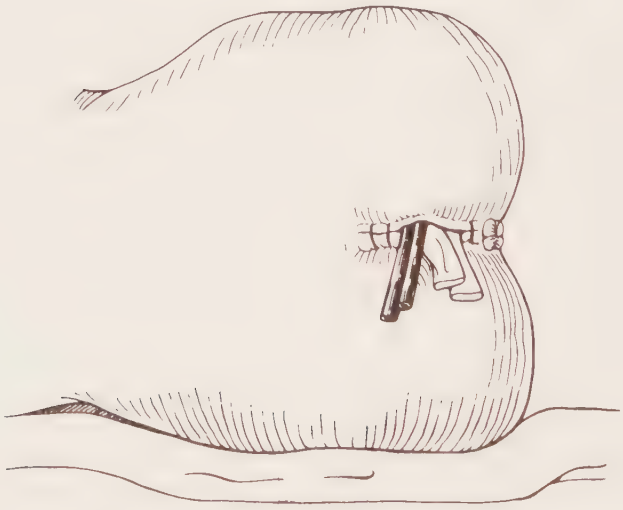


FIGURE 33.—Presacral drainage is best accomplished by direct vision through a posterior incision in a dependent position utilizing multiple soft rubber or sump drains.

distal segment must be irrigated at the time of initial wound surgery to assure removal of all fecal material. A loop colostomy does not meet these criteria and must be avoided.

Wound Closure

Closure of abdominal incisions should be carried out in anatomic layers. Since incidence of wound dehiscence in abdominal combat wounds is significant and since decreased atmospheric pressure during air evacuation flights may aggravate abdominal distention, the addition of retention sutures is a wise precaution. Retention sutures are mandatory, however, in patients who have had abdominal sepsis or repeated laparotomies. Wire or synthetic suture materials of adequate size should be used for this purpose. They should be placed and tied so as not to strangulate abdominal wall tissues.

Large abdominal wall wounds should be thoroughly debrided. If the resultant defect cannot be closed without tension by local soft tissue, Marlex mesh may be used even in the presence of contamination. Alternatively, a practical method for field use is to cover the defect with moist packs to prevent evisceration. Adhesions and granulations will form directly on the presenting abdominal contents, which may then be skin-grafted. Other foreign materials, including free fascia lata grafts, should not be used in the contaminated wound.

The cutaneous portion of the abdominal incision should be left open with a sterile dressing in place if there is a grossly contaminated wound. A delayed primary closure of the skin can then be carried out in 4 to 5 days without undue risk of infection.

Colostomy Closure

When the danger of peritonitis is past and recovery is assured, the colon is closed and its continuity reestablished by any desired technique. The procedure, which is elective, consists of appropriate resection of the bowel and intraperitoneal anastomosis. The safety of this procedure is predicated on ade-

quate preparation of the bowel, which includes thorough irrigation of the distal segment and the prophylactic use of antibiotics. This procedure is performed in hospitals of the communications zone and Zone of Interior and is usually possible in 4 to 6 weeks, although longer delays may be dictated by a continuing catabolic state or continuing peritoneal sepsis.

POSTOPERATIVE MANAGEMENT

The most important essential in the postoperative care of the patient with abdominal wounds is the necessity for an orderly, secure environment where careful observation and treatment may be continuous and may be performed by the same nurses and surgeons, when practicable. An attitude of confidence and reassurance on the part of the professional staff and other attendants is of great benefit in maintaining patient morale at this point.

Detailed postoperative care beginning with good nursing is essential to the successful management of abdominal wounds. That the reader is cognizant of the importance of sedation, monitoring vital signs, the value of deep breathing, and early ambulation is assumed.

Cardiorespiratory Care

In the early postoperative period, the patient who has had a serious abdominal wound with concomitant shock and who has been treated with large volumes of fluid and blood will require careful monitoring of central venous pressure, pO_2, pCO_2, and pH. Considerable intrapulmonary shunting because of alveolar hypoventilation may lead to unrecognized and severe hypoxemia. The failure to recognize this complication leads to unexpected cardiac arrest or the insidious development of the respiratory deficiency syndrome. Early signs and symptoms consist of increasing tachypnea with increased ventilatory effort, restlessness, nonproductive cough, or cough productive of clear, watery sputum. Cyanosis is not an early sign. The diagnosis is made by a high degree of suspicion and is confirmed

by demonstration of an increasing alveolar-arterial gradient when breathing 100 percent oxygen. The normal gradient is approximately 100 torr, and the development of a gradient of 300 torr or more is indicative of severe intrapulmonary shunting, causing large amounts of deoxygenated blood to return to the left heart. A shunt of over 50 percent is an indicator of poor prognosis. When the syndrome is recognized, treatment with volume-controlled ventilators using high airway pressures (up to 50–60 cm of water) and elevated end-expiratory pressures of 5–10 cm of water is indicated. Because of the progressively damaging effects of high oxygen concentration of the gas mixture, the lowest oxygen concentration (about 40 percent) is used, compatible with maintenance of reasonable oxygenation (approximately 70–80 torr arterial pO_2). Higher concentrations may, of course, become necessary to maintain this pO_2 if the syndrome progresses. Administration of intravenous albumin to increase intravascular osmotic pressure and the use of diuretics (20–40 mg furosemide) contribute to the decrease in pulmonary transudate and alleviation of the syndrome.

Thick sputum or tracheobronchial secretions or blood should be removed by frequent coughing, endotracheal aspirations, or bronchoscopy. Culture and smear of the sputum should be utilized to determine the necessity for specific antibiotic therapy.

Antibiotics

Parenteral, usually intravenous, antibiotics are given for 5 days. Penicillin, in doses of 20–40 million units, and usually a broad-spectrum antibiotic, such as tetracycline, at dose levels of 2–4 g per day are utilized. Such antibiotic usage, when prolonged, is complicated by emergence of resistant strains of unusual pathogens. The increase in infections with *Serratia marcescens* during the Vietnam conflict is such an example. For this reason, antibiotic administration beyond the fifth postoperative day should be employed upon strict indication and upon isolation of specific bacteria with definite sensitivity to the drugs chosen for treatment. The treatment of continuing sepsis includes careful examination of the abdomen, including roentgenologic study, to ascertain the need for reoperation

rather than the frenetic use of multiple antibiotic agents. Drainage of pus in such patients is more rewarding than any combination of antibiotics.

Blood Volume

Readjustment of the vascular system following severe injury and multiple transfusions must be monitored closely by determination of arterial blood pressure, central venous pressure, character of the pulse, skin capillary perfusion, and hourly urine output. A decreasing urine output and arterial pressure with a rising central venous pressure are particularly poor prognostic signs. In such a situation, isoproterenol may be useful and digoxin may be employed with some benefit after assurance that the serum potassium level is not low. The surgeon must be sensitive to the possible need for phlebotomy if the patient has been overtransfused or for further blood transfusions if blood volume is still low. If universal low-titer O blood has been given to a patient with A, B, or AB blood, the use of O blood should continue at this time and conversion to type-specific blood should take place only after about 3 weeks.

Fluid Administration

Accurate intake and output recordings are essential in guiding rational fluid therapy. A 40- to 100-ml hourly urine output of appropriate specific gravity is sought. Extrarenal loss of fluid is replaced with either normal saline or Ringer's lactate on a volume-for-volume basis. Insensible loss of 2,000 ml in a normal-sized man may be expected unless the ambient temperature is unusually high, the patient's temperature is elevated, or there are exposed moist body surfaces from which abnormal loss is occurring. Potassium replacement is started early if the urine output is adequate and if the patient is to remain on parenteral fluids more than 2 to 3 days. These patients lose large amounts of potassium from tissue breakdown, increased urinary excretion from the normal postoperative

adrenocorticoid effects, and from loss in gastric or intestinal fluids or from fistulas. An average daily maintenance in the postoperative period is 60–80 meq. The development of oliguric renal failure requires a sharp reduction in fluid administration (see ch. X). Hyperalimentation through a subclavian cannula must be started early if inability to take and retain adequate calories and proteins via the alimentary tract is anticipated. This early administration of high glucose-amino acid parenteral mixtures will circumvent the deleterious effects upon wound healing and susceptibility to infection brought on by a prolonged catabolic state.

Gastrointestinal Decompression

Nasogastric suction is continued until gastrointestinal function is reestablished. If the patient requires further operation and prolonged ileus is anticipated, the use of a tube gastrostomy or a long Baker intestinal tube via jejunostomy is particularly beneficial. The passage of flatus, the development of good peristaltic sounds, and the lessening requirements for intravenous fluids herald a return to normal of the gastrointestinal tract. If, for other reasons, the patient must be transferred by air to another hospital, the maintenance of a well-functioning tube is particularly important in prevention of abdominal distention, vomiting, and possible aspiration. Ideally, air evacuation should occur after a well-tolerated removal of the nasogastric tube and return of gastrointestinal tract function.

POSTOPERATIVE PERITONITIS

Peritonitis following penetrating wounds of the abdomen is the most frequent serious complication and leads to a significant delayed mortality. Complications which may contribute to the gravity of the situation are wound infection and wound dehiscence, persistent ileus, intestinal obstruction, atelectasis and pneumonia, intra-abdominal abscess formation, and stress ulceration.

Signs and Symptoms

The signs and symptoms of postoperative peritonitis are often so slight and insidious that they are regarded as part of the normal postoperative course. The temperature elevation may be no greater than would be expected after operation. Pain and rigidity are not notable, and distention is minimal. There may be only slight tenderness on palpation. Vomiting, masked by the use of nasogastric suction, may not be apparent until late. The patient usually appears anxious and restless, but his chief complaint may be little more than a feeling of distention. A persistently elevated pulse rate is an important sign.

Diagnosis

Because of the vagaries of postoperative peritonitis, repeated, careful examinations are necessary to detect its development. In the normal course of events, the abdomen becomes soft; peristalsis is restored; and temperature, pulse, and respiration return to normal within 72 to 96 hours after operation. When peritonitis develops, the abdomen is usually distended. Peristaltic sounds are infrequent and may be absent. Tenderness and rigidity are demonstrable. The patient is reluctant to change his position because movement increases his discomfort. Rectal examination may reveal diffuse tenderness and fullness. Pulse and temperature do not return to preoperative levels. Many of these signs and symptoms are often mistakenly attributed to the expected postoperative ileus or to delayed normal convalescence.

Leukocytosis may or may not occur, but nonsegmented polymorphonuclear leukocytes are always increased in number. Anemia is progressive.

Complications

Postoperative peritonitis almost invariably results in intra-abdominal abscess formation. The two most common locations are pelvic and subphrenic.

Pelvic abscess.—Pelvic abscesses usually are not difficult to diagnose. Surgery is indicated if there are no recession in the size of the abscess and no clinical improvement after a few days of correct supportive therapy. It is also indicated if the infection appears to be spreading and if signs and symptoms of rupture appear. If, on the other hand, the lesion appears to be localizing, operation may be delayed until the localizing process is complete.

Subphrenic abscess.—Subphrenic abscesses may develop as a result of any type of peritoneal contamination, though they most often occur after perforations of the stomach, the duodenum, or the right and the transverse colon.

Because of its anatomic location, recognition of a subphrenic abscess is sometimes difficult. Confirmation of the clinical suspicion depends upon a careful study of the progressive clinical signs and symptoms and evaluation of the progressive changes in serial fluoroscopic and roentgenologic examinations. A subphrenic abscess should be suspected whenever there is persistent and unexplained pyrexia after surgery and adequate supportive therapy.

If pain is present, it is referred to the posterior chest wall, the flank, the upper abdomen, or the shoulder. It sometimes can be produced by pressure thumping over the lower ribs posteriorly.

The liver is usually displaced downward, and its edge is palpable if the abscess is on the right side.

Roentgenologic and fluoroscopic studies are of great diagnostic value. Elevation of the diaphragm, which is almost always evident, is usually more pronounced on the affected side. The movement of the diaphragm, which is thickened, is restricted. The psoas shadow is usually indistinct on the involved side.

Pleural effusion with obliteration of the costophrenic angle is frequent. One should also search for displacement of the abdominal viscera and for collections of gas, which are often associated with fluid levels. The presence of gas in a subphrenic abscess usually results from infection by a gas-forming organism but may result from a communication between the abscess and the bowel, a bronchus, or even the exterior of the body.

Management

The treatment of peritonitis is both supportive and specific. Supportive care includes most of the measures discussed under postoperative management. Emphasis should be placed upon appropriate management of fluids and electrolytes, oxygen administration guided by blood gas determinations, effective antibiotic therapy guided by accurate culture and sensitivity data, blood transfusions to correct secondary anemia, and sedation to alleviate anxiety and pain.

In addition to pelvic and subphrenic abscesses, abscesses may occur in the lateral gutters and between loops of bowel. When localized abscesses are detected, appropriate posterior or lateral dependent drainage should be accomplished and maintained. Because of the multiple abscesses which frequently form, the abdomen should be formally explored for most abdominal abscesses which result from war wounds. Particularly if obstruction has been associated, the use of a Baker intestinal tube, inserted through a jejunostomy, is of benefit in splinting and decompressing the inflamed small intestine. Often the source of the sepsis is detected from this exploration; that is, anastomotic leak, missed abdominal injury, or further necrosis of tissue. These conditions can be corrected and appropriate dependent drainage established. Patients tolerate repeated operations poorly and every effort must be made to eliminate all sources of infection with the least number of surgical procedures. If the casualty has one obviously localized abscess which is well circumscribed without evidence of other infection, the abscess should be drained locally.

When adequate control of intra-abdominal sepsis is not established early, there is danger of septicemia with metastatic spread of infection to the lungs, brain, kidney, and liver. Once this occurs, the result is almost certain death. Pulmonary insufficiency, renal failure which is particularly refractory to treatment, and stress ulceration of the stomach and duodenum all have been linked to continuing sepsis.

EVACUATION

Experience shows that evacuation of casualties shortly after abdominal surgery carries a high mortality. Patients with wounds and injuries of the abdomen must be retained in the unit in which they have received their initial surgical care until complications have been controlled, bowel function has become normal, and the wound is healing. These requirements seldom can be met in fewer than 7 days.

CHAPTER XXIX

Reoperative Abdominal Surgery

This chapter considers abdominal reoperations performed because of complications arising in the early postinjury period. Indications for such operations are seldom well defined. These patients, because of the severity of their abdominal wounds and the high incidence of associated injuries, frequently present equivocal findings. With the development of sophisticated air evacuation, the occurrence of complications after departure from the hospital of original casualty care has become more common with earlier evacuation, that is, within the first 2 weeks. Records accompanying transferred casualties may lack sufficient details about the injury and the first operation to be helpful in subsequent evaluation. Given these circumstances, the surgeon must rely heavily on past experience for guidelines in reoperation of abdominal war wounds. The inherent problems in making a preoperative diagnosis in this most difficult group of patients should not deter an aggressive approach. This philosophy will prove much more rewarding than procrastination. Practical points gained from such experience follow.

TIME OF REOPERATION

The greatest number of reoperations for intra-abdominal complications occur in the first 3 weeks, with a peak incidence

from the fourth to the eighth day. This serves to identify that time frame in which the casualty should not be evacuated because frequent evaluation by experienced surgeons may not be available. If essential during this period, transfers that can be measured in hours rather than days are preferable. If a heavy patient load makes evacuation necessary, the least seriously injured patients should be selected rather than those with extensive intra-abdominal injury. This philosophy will allow for early diagnosis and treatment of complications in that group most likely to have them. The ideal status for evacuation of a patient with an abdominal wound is one wherein the patient is afebrile and alimentation has begun.

SPECIFIC REASONS FOR REOPERATIONS

Dehiscence

In addition to wound infection, two factors contribute to dehiscence: The failure to place retention sutures in war wounds, and the air evacuation of patients with postoperative ileus. The lowest incidence of dehiscence follows abdominal closure with retention sutures, 2 to 3 cm apart, through all layers in combination with closure of individual layers. Ileus is a factor in dehiscence because of pressure of distended bowel upon the abdominal wound. Bowel gas expands by 15 to 30 percent of sea level volume at the usual cabin pressure of evacuation aircraft. The avoidance, therefore, of evacuation when ileus exists is desirable. The use of nasogastric decompression minimizes this problem.

Retraction of Colostomy

Reoperation may become necessary in the early postoperative period because of retraction or necrosis of a colostomy or ileostomy. If the bowel has been exteriorized under tension, retraction may result. Tension becomes a problem when the bowel, usually a fixed portion of the bowel—that is, flexures or descending colon—is not adequately mobilized by liberal incision

in the lateral peritoneal reflections of the colon. Failure to suture the mesentery segment securely to the peritoneal also may favor retraction. Correction of this complication requires reoperation with performance of mobilization of the colon, which should have been performed at initial operation. Construction of another stoma in more proximal or retracted bowel under no tension is then possible. If easy deliverance of the bowel to the abdominal wall is not possible even after such mobilization due to inflammation shortening of the mesentery, performance of more proximal colostomy in a mobile portion of bowel, resecting bowel between the retracted colon and the new stoma, must be carried out. In the case of ileostomy, immediate maturation of the stoma by eversion and mucosa-to-skin suture with fine chromic catgut prevents retraction problems in this portion of the bowel. In addition, mesentery-to-peritoneum suture is necessary here as it is in the colon. If retraction of an ileostomy does occur, laparotomy is necessary to construct a new stoma in the fresh bowel slightly more proximal than the original site.

Missed Intra-abdominal Injury

Three factors have been found to influence the failure to identify and treat significant intra-abdominal injury: the adequacy of the operative incision, the necessity for complete systematic exploration, and the exploration by dissection of the hidden areas of the abdomen when indicated. The operative incision must be adequate in size as well as position. A generous midline incision is best for exposure and because of the facility with which it can be made and closed. Quadrant incisions are generally not so good unless the course of the wounding agent is known with absolute certainty, a situation which seldom prevails. A systematic exploration requires an adequate incision. An incision that admits only one of the operator's hands into the abdomen is inadequate for complete exploration. Change in location of certain intra-abdominal organs during changes in body position and respiration may be responsible for injuries distant from external wounds and is an additional reason for systematic, complete searching of all

organs. The most commonly overlooked injuries at celiotomy are those of the retroperitoneal structures, the fixed portions of the colon, and the viscera bordering the lesser sac. These areas can be inspected adequately only by intraoperative dissection and this should be done when there is any likelihood that injury to these organs has occurred.

Intra-abdominal injury can be overlooked when a missile penetrates the abdomen through an entrance site other than the anterior abdominal wall. When a patient who has undergone operative treatment of thigh, buttocks, chest, or flank wounds develops signs of peritonitis, a hidden abdominal wound must be suspected. Abdominal roentgenography may be of help by identifying a previously unrecognized intra-abdominal metallic fragment if one is present. This examination should be done in all such wounds to assist in early detection of these hidden wounds.

Stress Ulcer Hemorrhage

Upper gastrointestinal hemorrhage will occur most frequently in those casualties who are septic following intra-abdominal wounds, particularly when the colon is among the injured organs. For as yet unknown reasons, when sepsis is present without intra-abdominal injury, the incidence of stress bleeding is much less. The usual time of onset of bleeding is the 8th to the 12th day. Several modalities of nonoperative treatment have been employed in this problem. Those directed toward reducing visceral blood flow, such as the use of Pitressin and gastric cooling, have some usefulness. Although hyperacidity has not been found in the gastric aspirate of patients who subsequently bleed, antacids are frequently used empirically. In general, the success of controlling bleeding is correlated with the success of controlling sepsis. This is true whether operative or nonoperative means are used to control bleeding. Of the operations available for stress ulcer bleeding, none has emerged as an operation of choice, though vagotomy and pyloroplasty are those most commonly done. Subtotal gastrectomy may become necessary. Simple oversewing of ulcers usually fails to control bleeding.

Intestinal Obstruction

Mechanical intestinal obstruction is a late-occurring complication which usually develops within the first 2 weeks after injury. Early operative treatment has been employed with success, the use of long intestinal tubes having been less helpful. Adhesions and intraloop abscesses are the usual causes of obstruction in these cases. Intraoperative decompression and small bowel splinting to decrease the occurrence of subsequent obstruction may be effected by a long jejunostomy tube positioned at operation. It should be stressed that this complication occurs relatively late and should not be confused with prolonged ileus in the earlier postinjury period. Water-soluble radiopaque iodine compounds (Gastrografin) are employed to differentiate these two conditions. When administered orally, the contrast material fails to traverse the intestinal tract in mechanical obstruction. On the other hand, passage of the material through the intestinal tract over a few hours, evidenced by serial abdominal roentgenograms, often with initiation of bowel movements, aids in the diagnosis of the ileus.

Intra-abdominal Hemorrhage

Late hemorrhage in abdominal wounds is seen when an infectious process erodes a blood vessel of significant size. This occurs most often in the retroperitoneum when a hematoma is not explored. When resulting from a missile wound, the area is contaminated and eventual abscess or cellulitis reactivates bleeding by clot resolution or vessel erosion. Undetected wounds in the fixed portion of the colon, duodenum, pancreas, and retroperitoneum, with eventual infection, and hemorrhage from the lumbar venous plexus are most troublesome and may be fatal. Since such hemorrhage is usually profuse, operation, although mandatory, seldom saves the patient.

Intra-abdominal Abscesses

The drainage of abscesses accounts for the greatest number of reoperations in abdominal wounds. When the condition

presents in the relatively early postinjury period, it is most often associated with other complications, such as stress bleeding, fistula formation, and intestinal obstruction. Abscesses may also be chronic and present much later with low-grade fever and inanition.

Treatment involves evacuating the abscess, collapsing the cavity, and preventing recurrence. High-suction sump drains, using irrigation with saline or antibiotic solutions, help to achieve these ends. If the cavity is not well formed, irrigation may spread organisms to other intra-abdominal sites. Judgment, therefore, must be exercised regarding the use and volume of irrigating solutions. Drains should be dependently positioned to achieve the maximum effect of gravity.

Large Abdominal Wall Defects

When a considerable portion of the abdominal wall has been lost as a result of a wound or necessary debridement, the surgeon must consider the effects of initial treatment on the subsequent course. If a primary closure is attempted, strangulation of tissue by undue tension may cause necrosis of wound edges. The tight closure leads to limitation of diaphragmatic excursion and respiratory compromise. If the small intestine is allowed to become the base of a granulating wound, fistula formation and intestinal obstruction may result. Both of these situations may require reoperation. The most successful form of treatment, at initial operation or reoperation, has been the insertion of a Marlex mesh prosthesis, sewn to the undersurface of the remaining viable abdominal wall. While it is becoming encased in granulation tissue, the mesh should be covered with a dressing soaked in physiologic saline containing an antibiotic. Neomycin or other broad-spectrum antibiotics are acceptable. Once the base of the wound is covered by healthy granulation tissue, it can be covered by a split-thickness skin graft or a sliding pedicle graft. When Marlex is unavailable, the wound should be temporarily covered by a nonadherent synthetic material, such as parachute nylon, held in position under large horizontal mattress sutures to permit safe evacuation.

Fistulas

A well-formed fistula, regardless of source, may be treated by high-volume suction. Closure eventually occurs when distal obstruction is not present. When the course of the fistula is circuitous or unknown, or when it is associated with abscesses or peritonitis, a direct operative attack is indicated. The choice of operation depends on the organ involved. Closure with adequate drainage, resection, exteriorization, repair, and proximal diversion, singly or in combination, should be employed as the local situation dictates.

CHAPTER XXX

Wounds and Injuries of the Genitourinary Tract

Genitourinary tract injuries in a combat zone constitute approximately 5 percent of the total injuries encountered. With the exception of the external genitalia, these wounds invariably will be associated with serious visceral injury and, as a result, are generally managed in areas where there are major surgical and roentgenographic capabilities. The treatment of urologic injuries does not vary from established surgical principles; that is, bleeding control, adequate debridement, and drainage. In contrast to intraperitoneal injuries, preoperative evaluation, utilizing appropriate urographic diagnostic procedures, is simpler and more expedient than an extensive retroperitoneal exploration at the time of laparotomy and particularly rewarding when an unsuspected injury, anomaly, or absence is disclosed.

WOUNDS OF THE KIDNEY

Renal injuries may be classified as open or closed. All open renal injuries require surgical intervention; closed injuries are usually best treated nonoperatively unless continuing blood loss prompts an operation. The routine of investigation is as follows:

1. A complete physical examination is carried out, to permit evaluation of the patient's status and, if other injuries are present, to establish priority of treatment.

2. Urinalysis is done, to confirm or exclude hematuria.
3. Scout films of the abdomen are made.
4. Intravenous or retrograde pyelography is carried out when facilities are available. The presence and function of the opposite kidney are determined before surgery is undertaken. This precaution should never be omitted for it may determine the kind of surgery to be performed.

Nonoperative treatment consists of strict bed rest and fluid and blood replacement. Operative treatment consists of local debridement and suture, total nephrectomy, or, rarely, partial nephrectomy. An abdominal incision is used in initial operation in open wounds involving the kidney. The single most important consideration at the time of exploration is control of the vascular pedicle before opening the perirenal tissues and releasing the hemostatic tamponade. Drainage of the renal fossa is mandatory. Nephrostomy is indicated after partial nephrectomy.

WOUNDS OF THE URETER

Ureteral injuries are rare and are frequently overlooked. The diagnosis is made only if the possibility of such an injury is considered in all cases of retroperitoneal hematoma and injuries of the fixed portions of the colon, the duodenum, and the spleen. Complete transection of the ureter necessitates reapproximation of the edges with interrupted 4–0 or 5–0 chromic catgut sutures which do not penetrate the mucosa. Proximal drainage must be instituted either by nephrostomy or ureterostomy using a ureteral catheter or T-tube. Ureteral injuries near the bladder can be treated by reimplantation of the ureter into the bladder. Injuries from high-velocity missiles may involve considerable loss of ureteral substance. This loss may be obvious or it may be a segment of poorly defined devitalized tissue. Adequate drainage at the site of ureteral injury is always required.

WOUNDS OF THE BLADDER

Bladder wounds are common and always should be considered in cases of pelvic trauma. Bladder tears may be intra-

peritoneal or extraperitoneal. In either case, the treatment consists of closure of the tear or perforation with one or two layers of absorbable suture, not penetrating the mucosa, retropubic drainage, and suprapubic cystostomy with a large bore tube capable of evacuating clots. The diagnosis of penetrating bladder injury or tear is best made by use of cystography. The technique consists of filling the bladder by gravity with radiopaque fluid placed 20–30 cm above the level of the abdomen, taking a film, evacuating the bladder with the catheter, and obtaining a repeat film. Frequently, the last film taken will disclose small tears and resultant extravasation which is obscured by the large bolus of contrast agent. Penetrating injuries of a hollow viscus should have an even number of wounds corresponding to the sites of entrance and exit wounds.

WOUNDS OF THE URETHRA

No other urologic injury has the potential for long term morbidity as does urethral injury, and frequently initial treatment determines what disability the patient will incur in the future. The urethra is divided into an anterior and a posterior (prostatic) part by the urogenital diaphragm. The cardinal sign of anterior urethral injury is the presence of blood at the external urethral meatus. The diagnosis is confirmed by retrograde urethrography. Minor urethral injuries can be treated by splinting with a 16 or 18 Fr catheter for 10 days. Partial urethral injuries are best treated by suprapubic cystostomy. Marsupialization—that is, suturing skin edges to the cut edges of the urethra—is excellent in certain cases. This is followed at a later stage by closure of the marsupialized area to reestablish urethral continuity. Complete urethral transection should be treated by suprapubic cystostomy and reapproximation of the urethral edges with fine chromic catgut. There should be no tension on the suture line. Additional urethral length can be obtained by dissecting the urethra and corpus spongiosum from the corpora cavernosa from the corona to bulb if necessary. It is also advantageous to spatulate the urethral ends to avoid a circumferential constricting scar and to use as few sutures as possible to obtain anatomic but not watertight approximation. Drainage of the injury site is essential.

Posterior urethral injuries are almost always associated with pelvic fractures. Rectal examination reveals the prostate to have been avulsed at the apex. Once the condition is diagnosed, the patient is instructed not to try to void and an immediate attempt is made to pass a small Foley catheter. This, on occasion, is successful, in which case the catheter is left in place and no further immediate therapy is indicated. More often, however, it is necessary to open the retropubic space and the bladder. A Foley catheter is passed per urethram, and a Robinson catheter passed through the bladder neck. Both catheters are identified in the space of Retzius and tied together, and the Foley is delivered into the bladder. The balloon is inflated to at least 30 ml, more if possible; the catheter is placed on gentle traction and anatomic continuity is restored. If possible, a traction suture of No. 1 chromic catgut should be passed through the prostate and out of the perineum, where it is tied over a gauze roll. This prevents the prostate from riding up in case of balloon breakage or slippage through the prostatic urethra. It is important to add the safety factor: a suture through the eye of the Foley catheter is brought through the suprapubic cystostomy tube, which will facilitate its replacement if necessary. Adequate drainage of the prevesicle space is mandatory. Colostomy should be considered if a large open perineal wound is present.

WOUNDS OF THE EXTERNAL GENITALIA

The management of wounds of the penis, scrotum, testes, and spermatic cord consists of control of hemorrhage; debridement, which should always be as conservative as possible; and repair as early as possible, to prevent deformity.

In injuries of the penis, tears in Buck's fascia should be sutured. When denudation has been extensive, the penis may be placed in a scrotal tunnel until plastic repair can be carried out in an appropriately equipped facility.

The scrotum has a good blood supply, and extensive debridement is therefore not necessary. In complete avulsion, the testes can be placed in protective pockets in the thighs.

In testicular injuries, it is essential that all tissue possible be conserved. Herniated parenchymal tissue should be replaced and the tunica albuginea closed by mattress sutures. The testicle is placed in the scrotum or in a protective pocket in some adjacent structure. A testicle should never be resected unless it is hopelessly damaged and its blood supply destroyed.

CHAPTER XXXI

Wounds and Injuries of the Hand

A hand injury is a matter of the greatest importance. Aside from his brain, a man's hand is his most important nonvital endowment. With it, he masters his environment for survival and happiness. Without it, he becomes helpless and his tools become useless. The instruments of war are without value if there are no usable hands to operate them.

The hand is constantly subject to serious injury, and even minor injuries may be incapacitating. Fortunately, the hand has remarkable recuperative powers; even if it is crippled, it can be trained to compensate for much of its lost function. However, the injured hand should never be taken lightly, even when trauma to it seems comparatively minor. Even when the loss of tissue and the resulting loss of function are severe, much dysfunction can be prevented in both major and minor injuries by proper early care. Prevention of hand injuries and prompt and effective care following a hand injury are of prime importance in the medical military responsibility.

Injuries of the hand seldom, in themselves, cause fatalities or even shock. A casualty who is in a state of shock when first seen, therefore, should be investigated for other wounds which may be responsible for it. These wounds to more vital structures should be managed as necessary before attending to the wound of the hand. Delay in treatment of the hand injury is always justified if hemorrhage has been checked and the injured member is protected from further injuries by dressings and immobilization.

Knowledge of the functional anatomy of the hand is essential for proper surgical repair and restoration.

CARE IN THE DIVISION AREA

Care of the wounded hand in the division area is limited to control of hemorrhage and to immobilization. No attempt is made to repair the damage until X-rays have been taken and there is sufficient time for orderly evaluation of the injury to each element in the intricate mechanism.

In the interim, indicated supportive therapy is begun, and the casualty is evacuated to an installation with surgical facilities, with the hand splinted in the position of function (fig. 34) and supported by a sling.

If delay in evacuation is inevitable, the following additional measures are carried out:

1. Wristwatches and rings are removed. The hand is thoroughly cleansed with soap and water, and the fingernails, which are usually black with dirt, are clipped and cleaned.

2. Shreds of obviously dead tissue are excised. Amputation of digits is rarely indicated. If amputation is necessary, it is done later, after the opportunity for more careful evaluation and planning becomes available.

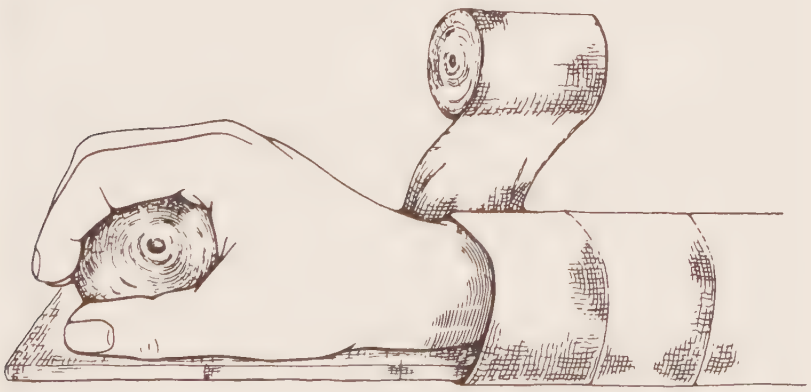


FIGURE 34.—Immobilization of injured hand and forearm in position of anatomic function.

3. Oozing is controlled by the application of a generous gauze dressing. Petrolatum-impregnated gauze and grease in any form are avoided.
4. The hand is immobilized in the position of function.

INITIAL WOUND SURGERY

Anesthesia

Local infiltration does not provide satisfactory anesthesia for surgery in open wounds of the hand. Both nerve block (conduction) and general anesthesia are satisfactory and either may be used as the situation dictates. Adrenalin is never used with local anesthesia in surgery of the hand and fingers because of the risk of precipitating vasospasm and subsequent gangrene.

Debridement

For a successful operation, the surgeon requires an assistant, good light, and enough time. He should operate seated, with the draped extremity extended and the hand resting on a suitable support. Hand surgery also requires proper instruments and sutures. Necessary instruments include fine tissue forceps, skin hooks, straight and curved ophthalmic scissors, small knife blades, and fine needles. Fine plain catgut is used for deep suture.

Cleansing of the surgical field before surgery is most important. The hand of the wounded soldier is filthy, and attention to detail at the time of the initial cleansing is invaluable in diminishing sepsis. A meticulous shave followed by trimming and cleansing of the fingernails and a circumferential scrub from the tip of the fingers to the tourniquet are required.

Hemostasis must be complete. It is accomplished with the aid of intermittent application of the tourniquet, which is never kept functioning for more than 1 hour at a time. The tourniquet is always released before application of the final dressing. When it is released, fine bleeding points are identified and ligated as necessary. When injured, either the radial or the

ulnar artery usually can be safely ligated, if necessary, since both have rich terminal anastomoses. Both arteries, however, should never be ligated.

Trimming of the wound edges should not be routinely carried out. The removal of a few millimeters of normal skin may result in contracture and frequently necessitate skin grafting. Contused skin or skin tattooed with dirt may be used at times for delayed closure, and so none should be excised routinely.

The deep structures of the hand should be explored thoroughly to allow adequate debridement and to determine the extent of injury to structures noted to be damaged in the preoperative examination. It is necessary during the operative procedure to think anatomically so as not to damage previously undamaged structures. The carpal tunnel may be opened to visualize and protect the median nerve branches during debridement. Additionally, this incision of the transverse carpal ligament decompresses the median nerve and improves tendon function in the severely damaged hand. In certain injuries associated with massive swelling of the hand, decompression of the intrinsic muscles may be indicated. Removal of the intermetacarpal fascia through small dorsal incisions will reduce the possibility of developing intrinsic contracture.

Dead muscle, tissue, clots, readily accessible foreign bodies, and other debris are removed. Bone fragments which are not grossly contaminated are preserved. Severely damaged and useless tendons should be excised. Every bit of viable tissue should be preserved. During these procedures, the wound is frequently irrigated with physiologic salt solution.

Only digits which are irretrievably damaged are amputated. Amputation of the thumb is a last resort, performed only after repeated evaluation. It is sometimes possible to save skin from a finger which must be amputated, to provide later coverage for the remainder of the hand. In digital amputations in which this seems a possibility, the tendons should be removed with the bone, but the digital vessels and nerves should be retained. Tendon repair, including tendon grafts, is not the responsibility of the forward surgeon.

Nerves which are traumatically divided are usually disrupted over a considerable distance and should not be primarily re-

paired. However, nerves which can be approximated in relatively healthy tissue without any tension should be approximated with one or two sutures of nonabsorbable suture material. This will prevent retraction and enhance the technique of a future neurorrhaphy without undue traction or tension on the involved nerve. Digital nerves, in particular, should be repaired primarily with the expectation of avoiding a painful neuroma.

WOUND CLOSURE

In most cases, it is best to delay closure of the wound for several days after the initial wound surgery. In this way, one can be sure that the wound is free of sepsis and necrotic tissue at the time of closure. Although it is possible to perform primary closure in certain wounds of the hand, the possibility of deep sepsis and wound breakdown does not justify primary closure in the combat situation.

At the time of wound closure—that is, within 3 to 5 days—skeletal stability may be achieved with small Kirschner wires in unstable fractures or dislocations. Stability thus achieved results in a hand which can be actively moved in the postwound period and also prevents the development of later deformity. This internal fixation should not be used unless the wound is free of infection and ready for closure.

Dressing

The dressing consists of well-fluffed gauze, applied evenly and snugly over a layer of fine-mesh gauze. Petrolatum-impregnated gauze must not be used. The deeper parts of the wound must not be plugged. The fingers are spread without tension, with the thumb in opposition. Padding is placed between the fingers. An attempt is made to aline all fractures while applying the dressing.

The dressing should cover the entire wound but should not constrict it. It is reinforced with layers of sterile absorbent cotton covered by a firm pressure bandage. Unaffected digits

are left free to move, and, whenever possible, the tips of all fingers are left bare so that they can be inspected regularly to determine the adequacy of the blood supply.

Splinting

The hand is placed in the position of function; that is, it is supported on a molded volar plaster splint, with the wrist dorsiflexed approximately 30 degrees, the fingers flexed at all joints (30 to 40 degrees), and the thumb slightly flexed and rotated opposite the palm. This is the position in which the hand would hold a glass. Only the injured fingers are splinted. The others are left free to move.

Postoperative Management

After operation, the hand and arm are elevated, by a sling if the patient is ambulatory or across the chest if he must be recumbent or is evacuated by litter. Movement of all uninvolved joints is insisted on.

APPENDIX A

Glossary of Drugs With National Nomenclatures

ANESTHETICS

United States	Germany	Netherlands	France
Chloroprocaine N.F.	.....	Chloroprocaine.	
Cocaine hydrochloride U.S.P.	Cocainhydrochlorid DAB 7..	Cocaine hydrochloride ..	Cocaine chlorhydrate.
Cyclopropane U.S.P.	.....	Cyclopentaan	Cyclopropane.
Droperidol	Dehydrobenzperidol	Droperidol	Haldol.
Ether U.S.P.	Aether pro narcosi DAB 7..	Ether U.S.P.	Ether pur pour anesthesie.
Halothane U.S.P.	Halothan	Halothaan	Halothane.
Lidocaine hydrochloride U.S.P.	Lidocainii hydrochloridum Ph. Int.	Lidocaine hydrochloride.	Xylocaine.
Mepivacaine hydrochloride N.F.	Scandicain	Mepivacaine hydrochloride.	
Nitrous oxide U.S.P.	Distickstoffoxide, Lachgas ..	Lachgas, distikstofoxide..	Protoxyde d'azote.
Prilocaine hydrochloride N.F.	.....	Prilocaine hydrochloride.	
Procaine hydrochloride U.S.P.	Procainhydrochlorid DAB 7.	Procaine hydrochloride..	Lignocaine.
Proparacaine hydrochloride U.S.P.	.....	Proparacaine hydrochloride, proxymetacaine.	
Tetracaine hydrochloride U.S.P.	Tetracainhydrochlorid DAB 7.	Tetracaine hydrochloride	Tetracaine.
Thiopental sodium U.S.P...	Trapanal	Thiopental-natrium	Penthiobarbital injectable.

MUSCLE RELAXANTS

Curare	.....	Curare.	
Pancuronium bromide	.....	Pancuronium bromide.	
Succinylcholine U.S.P.	Suxamethonium chloridum Ph.Int.	Succinylcholine	Succinylcholine.
Tubocurarine chloride U.S.P.	D-Tubocurarini chloridum Ph.Int.	Tubocurarine chloride ..	Tubocurarine.

NARCOTICS

Codeine phosphate U.S.P. ..	Codeinphosphate DAB 7...	Codeine fosfaat	Codeine.
Meperidine hydrochloride U.S.P.	Pethidini hydrochloridum Ph.Int.	Pethidine hydrochloride.	Pethidine chlorhydrate.
Methadone hydrochloride U.S.P.	Methadoni hydrochloridum Ph.Int.	Methadon hydrochloride.	
Morphine sulfate U.S.P.	Morphini sulfas Ph.Int.	Morfine sulfaat	Morphine.
Papaverine hydrochloride N.F.	Papaverinhydrochlorid DAB 7.	Papaverine hydrochloride	Papaverine.

HYPNOTICS

Amobarbital U.S.P.	Amobarbitalum Ph.Int.	Amobarbital	Amobarbital.
Paraldehyde U.S.P.	Paraldehyd DAB 7	Paraldehyde	Paraldehyde.
Pentobarbital U.S.P.	Neodorm	Pentobarbital	Nembutal.
Phenobarbital U.S.P.	Phenylaethylbarbitursäure DAB 7.	Fenobarbital	Phenobarbital.
Secobarbital U.S.P.	.....	Secobarbital	Secobarbital.

ANTIBIOTICS

United States	Germany	Netherlands	France
Ampicillin U.S.P.	Amblosin, Binotal	Ampicilline	Ampicilline.
Bacitracin U.S.P.	Bacitracin	Bacitracine	Bacitracine.
Carbenicillin	Anabactyl	Carbenicilline.	
Cephaloridine		Cefaloridine	Cephaloridine.
Cephalothin U.S.P.	Cefalotin	Cefalotine	Cephalotine.
Chloramphenicol U.S.P.	Chloramphenicol DAB 7	Chlooramfenicol	Chloramphenicol.
Colistin N.F.	Colistin	Colistine	Colistine.
Erythromycin U.S.P.	Erythromycinum Ph.Int.	Erythromycine	Erythromycine.
Gentamicin sulfate U.S.P.	Refobacin	Gentamicine sulfaat	Gentamycine.
Kanamycin U.S.P.	Kanmytrex, Kanamycin, Resistomycin.	Kanamycine	Kanamycine.
Lincomycin U.S.P.	Cillimycin	Lincomycine.	
Methicillin U.S.P.	Cinopenil	Methicilline	Methicilline.
Neomycin sulfate U.S.P.	Neomycin, Bykomycin	Neomycine sulfaat	Neomycine.
Oxacillin U.S.P.	Cryptocillin, Stapenor	Oxacilline	Oxacilline.
Polymyxin B sulfate U.S.P.	Polymyxini B-sulfas Ph.Int.	Polymyxine B sulfaat	Polymyxine B.
Potassium penicillin G, U.S.P.	Penicillin G-Kalium DAB 7.	Kalium penicilline B	Penicilline.
Potassium phenoxymethyl penicillin U.S.P.	Beromycin, Immunocillin, Isocillin, Aracil.	Kalium fenoxymethyl penicilline.	
Streptomycin sulfate U.S.P.	Streptomycinsulfat DAB 7.	Streptomycine sulfaat	Streptomycine sulfate.
Tetracycline U.S.P.	Tetracyclinum Ph.Int.	Tetracycline	Oxytetracycline.

DRUGS AFFECTING SYMPATHETIC NERVOUS SYSTEM AND NERVE ENDINGS

Atropine sulfate U.S.P.	Atropinsulfat DAB 7.....	Atropine sulfaat	Atropine sulfate.
Cyclopentolate hydrochloride U.S.P.	....	Cyclopentolaat hydrochloride.	
Epinephrine U.S.P.	Adrenalin DAB 7.....	Adrenaline	Adrenaline.
Homatropine hydrobromide U.S.P.	Homatropinhydrobromid DAB 7.	Homatropine hydrobromide.	Homatropine bromhydrate.
Isoproterenol U.S.P.	Isoprenalini hydrochloridum Ph.Int.	Isoprenaline	Isuprel, isoprenaline.
Levarterenol bitartrate U.S.P.	Noradrenalinhydrogentartrat DAB 7.	Levarterenol bitartraat .	Noradrenaline.
Physostigmine salicylate U.S.P.	Physostigminsalicylat DAB 7	Fysostigmine salicyclaat..	
Pralidoxime chloride U.S.P..	Pralidoximi methiodidum Ph.Int.	Pralidoxime chloride ...	Pralidoxime.
Scopolamine U.S.P.	Scopolaminhydrobromid DAB 7.	Scopolamine U.S.P.	Scopolamine.

ELECTROLYTES

Calcium gluconate injection U.S.P.	Calcium gluconicum 10% ..	Calcium gluconaat injectie.	Calcium levulinate.
Dextrose injection U.S.P. ...	Traubenzuckerlösung 10%..	Glucose injectie	Solution injectable de glucose, isotonique.
Lactated Ringer's injection U.S.P.	Ringer-Lactat-Lösung	Ringer lactaat injectie ..	Lactate de calcium.
Ringer's injection U.S.P. ...	Ringer-Lösung	Ringer injectie	Solution de Ringer.
Sodium chloride injection U.S.P.	Natriumchloridlösung, isotonisch, pyrogenfrei, steril (DAB 7).	Natrium chloride injectie.	Solution injectable de chlorure de sodium, isotonique.

PLASMA EXPANDERS

United States	Germany	Netherlands	France
Normal human serum albumin U.S.P.	Humanalbumin 20%	Normal humaan albumine uit serum.	Albumine humaine.
Plasma protein fraction U.S.P.	PPL, Humanalbumin 5%...	Protein fractie uit plasma.	Gamma globuline.

MISCELLANEOUS DRUGS

Acetylsalicyclic acid U.S.P. ..	Acetylsalicylsäure DAB 7 ...	Acetylsalicyl zuur	Acetylsalicylique acide.
Amyl nitrite N.F.	.....	Amyl nitriet	Nitrite d'amyle.
Chlorpromazine U.S.P.	Chlorpromazini hydrochloridum Ph.Int.	Chloor promazine	Chlorpromazine.
Digitalis U.S.P.	Digitalisblätter DAB 7	Digitalis	Digitaline.
Digoxin U.S.P.	Digoxinum Ph.Int.	Digoxine.	
Ethacrynic acid U.S.P.	.....	Ethacryne zuur.	
Furosemide U.S.P.	Lasix	Furosemide	Furosemide-Lasilix.
Hydrocortisone sodium succinate injection U.S.P.	Hydrocortisonhemisuccinat .	Hydrocortison natrium succinaat injectie.	Hydrocortisone hemisuccinate.
Mafenide	Marfanil	Mafenide.	
Mannitol U.S.P.	D-Mannit	Mannitol.	
Oxygen U.S.P.	Sauerstoff	Zuurstof	Oxygene.
Petrolatum U.S.P.	Paraffinum solidum DAB 6, Paraffinum durum DAB 7.	Vaseline	Vaseline.
Probenecid U.S.P.	Benemid	Probenecide.	
Silver nitrate U.S.P.	Silbernitrat DAB 7	Zilvernitraat	Nitrate d'argent.
Sodium nitrite U.S.P.	Natriumnitrit DAB 7.....	Natrium nitriet	Nitrite de sodium.
Sodium polysterene sulfonate U.S.P.	Kationen-Austauscherharz ..	Natrium polystyreen sulfonaat.	
Sodium thiosulfate U.S.P. ..	Natriumthiosulfat DAB 7...	Natrium thiosulfaat	Hyposulfite de sodium.

VACCINES AND ANTITOXINS

Gas gangrene antitoxin, pentavalent.	Gasoedem-Antitoxin, polyvalent.	Gas gangreen—antitoxine pentavalent.	Serum antigangreneux polyvalent.
Tetanus immune globulin (human) U.S.P.	Tetanus-Immunglobulin ...	Tetanus-immuno globuline (menselijk).	
Tetanus toxoid U.S.P.	Tetatoxoid	Tetanus vaccin	Vaccin antitetanique.

ANTISEPTICS

Alcohol U.S.P.	Aethanol DAB 7	Alcohol	Alcool ethylique.
Hexachlorophene U.S.P.	Hexachlorophen WHO	Hexachlorophene	Exophene.

APPENDIX B

Useful Tables

TABLE 1.—*Atomic weights, valences, and equivalent weights of certain elements*

Element	Atomic weight	Valence	Equivalent weight
Sodium	23.0	1	23.0
Potassium	39.0	1	39.0
Magnesium	24.0	2	12.0
Calcium	40.0	2	20.0
Chlorine	35.5	1	35.5
Phosphorus	31.0	3	10.3
Sulfur	32.0	2	16.0

TABLE 2.—*Milliequivalent per gram of certain elements and compounds*

Element or compound	Milli-equivalent	Element or compound	Milli-equivalent
Sodium	43.5	Potassium chloride:	
Potassium	26.0	Potassium	13.5
Magnesium	85.0	Chloride	13.5
Calcium	50.0	Sodium lactate:	
Chloride	29.0	Sodium	9.0
Sodium chloride:		Lactate	9.0
Sodium	17.0		
Chloride	17.0		

TABLE 3.—*Normal range of concentration of serum constituents*

Constituent	Concentration per 100 milliliters	Milliequivalent per liter
Sodium ..... milligram	310–340	135–148
Potassium ..... do	13.6–20.7	3.5–5.3
Magnesium ..... do	1.8–3.0	1.5–2.5
Calcium ..... do	9.6–11.0	4.8–5.4
Chloride ..... do	348–383	98–108
Urea nitrogen ..... do	8–26	
Carbon dioxide	101–132	23–30 mM
Total protein ..... gram	6.0–8.2	
Albumin ..... do	3.8–5.0	
Globulin ..... do	2.3–3.5	

TABLE 4.—*Normal range of concentration of whole blood gases and pH values*

Constituent	Concentration
pO_2—Arterial	90–100 torr [1]
Venous	30 torr
pCO_2—Arterial	35–46 torr
Venous	38–49 torr
pH	7.35–7.45

[1] One torr = 1 mm Hg at sea level.

TABLE 5.—*Equivalent United States and imperial weights and measures*

Unit of measurement	Abbreviation	United States measure	Imperial measure
1 milligram	mg	0.015432 grain	0.015432 grain.
1 gram	g	15.432 grains	15.432 grains.
1 kilogram	kg	35.274 ounces (avoirdupois)	35.274 ounces (avoirdupois)
		32.150 ounces (apothecary)	35.274 ounces (apothecary).
1 grain	gr	0.0648 gram	0.0648 gram.
		64.8 milligrams	64.8 milligrams.
480 grains	gr	31.1035 grams	31.1035 grams.
1 ounce	oz	437.5 grains	437.5 grains.
		28.350 grams	28.350 grams.
1 milliliter	ml	16.23 minims	16.894 minims.
1 liter	l	33.814 fluid ounces	35.196 fluid ounces.
1 minim	min	0.0616 milliliter	0.0592 milliliter.
1 fluid ounce	fl oz	29.573 milliliters	28.412 milliliters.
1 pint[1]	pt	473.17 milliliters	568.25 milliliters.

[1] Sixteen fluid ounces, U.S. measure; 20 fluid ounces, imperial measure.

TABLE 6.—*Approximate equivalent metric and imperial doses*

Milliliters	Minims	Grams	Grains	Milligrams	Grains
0.1	1½	0.1	1½	0.1	1/600
0.12	2	0.12	2	0.125	1/480
0.15	2½	0.15	2½	0.25	1/240
0.2	3	0.2	3	0.3	1/200
0.25	4	0.25	4	0.5	1/120
0.3	5	0.3	5	0.6	1/100
0.4	6	0.4	6	1	1/60
0.5	8	0.5	8	1.5	1/40
0.6	10	0.6	10	2	1/30
1	15	1	15	2.5	1/24
1.3	20	1.3	20	3	1/20
2	30	2	30	5	1/12
3	45	3	45	8	1/8
4	60	4	60	9	3/20
5	75	5	75	10	1/6
6	90	6	90	12	1/5
8	120	8	120	16	1/4
10	150	10	150	20	1/3
15	225	15	225	25	2/5
20	300	20	300	30	1/2
25	375	25	375	50	3/4
				60	1
				75	1¼

TABLE 7.—*Equivalent avoirdupois and metric weights*

Pounds	Kilograms	Pounds	Kilograms
100	45.359	155	70.306
105	47.627	160	72.574
110	49.895	165	74.842
115	52.163	170	77.110
120	54.431	175	79.378
125	56.698	180	81.646
130	58.966	185	83.914
135	61.234	190	86.182
140	63.502	195	88.450
145	65.770	200	90.718
150	68.038		

TABLE 8.—*Equivalents of centigrade and Fahrenheit thermometric scales*

Degrees C	Degrees F	Degrees C	Degrees F	Degrees C	Degrees F
−10	14.0	31	87.8	72	161.6
−9	15.8	32	89.6	73	163.4
−8	17.6	33	91.4	74	165.2
−7	19.4	34	93.2	75	167.0
−6	21.2	35	95.0	76	168.8
−5	23.0	36	96.8	77	170.6
−4	24.8	37	98.6	78	172.4
−3	26.6	38	100.4	79	174.2
−2	28.4	39	102.2	80	176.0
−1	30.2	40	104.0	81	177.8
0	32.0	41	105.8	82	179.6
1	33.8	42	107.6	83	181.4
2	35.6	43	109.4	84	183.2
3	37.4	44	111.2	85	185.0
4	39.2	45	113.0	86	186.8
5	41.0	46	114.8	87	188.6
6	42.8	47	116.6	88	190.4
7	44.6	48	118.4	89	192.2
8	46.4	49	120.2	90	194.0
9	48.2	50	122.0	91	195.8
10	50.0	51	123.8	92	197.6
11	51.8	52	125.6	93	199.4
12	53.6	53	127.4	94	201.2
13	55.4	54	129.2	95	203.0
14	57.2	55	131.0	96	204.8
15	59.0	56	132.8	97	206.6
16	60.8	57	134.6	98	208.4
17	62.6	58	136.4	99	210.2
18	64.4	59	138.2	100	212.0
19	66.2	60	140.0	101	213.8
20	68.0	61	141.8	102	215.6
21	69.8	62	143.6	103	217.4
22	71.6	63	145.4	104	219.2
23	73.4	64	147.2	105	221.0
24	75.2	65	149.0	106	222.8
25	77.0	66	150.8	107	224.6
26	78.8	67	152.6	108	226.4
27	80.6	68	154.4	109	228.2
28	82.4	69	156.2	110	230.0
29	84.2	70	158.0		
30	86.0	71	159.8		

Index

Abdomen:
  blast injuries, 52–54
  distention in spinal cord injuries, 262–263
  exploration, 191
  missed injury, 342–343
  reoperative surgery, 340–346
  roentgenographic examination, 85
  surgery. *See* Laparotomy.
  vascular injury in, 208, 217
  wound closure in, 331
  wounds, 132, 183, 232, 249, 261, 315–339
Abdominal wall:
  blast injury to, 41
  defects, 345
Abscesses:
  abdominal, 338, 344–345
  formation in maxillofacial wounds, 267
  gas, 124
  in peritonitis, 336–337
  intestinal, 344
  retroperitoneal, 123
  with fistulas, 346
Accidents, stopping breathing, 167
Acidosis, 33, 102, 110, 114
Adhesions, intestinal, 344
Adrenalin. *See* Epinephrine.
Aerobacter, in war wounds, 122
Aero-otitis, 293–294
Age, role in cold injury, 39
Aidman, medical, duties, 156, 180
Air blast, 48–49
Aircraft:
  for rapid patient movement, 159–160
  pressurization, 162
Aircrews, 37
Air evacuation, 3, 29, 155, 159–164, 253, 341
  See also Evacuation: Helicopter evacuation.
Air leak, 311
Airway:
  artificial, respiration using, 172
Airway—Continued
  blockage, 269
  emergencies, 302
  establishment, 84, 166
  postoperative care, 195
Airway maintenance, in—
  burns, 18, 22, 23, 26, 34
  chest wounds, 308
  head wounds, 249
  laryngotracheal injury, 297
  maxillofacial wounds, 183, 267, 268
  neck injuries, 298
  respiratory obstruction, 167
  shock, 97
  surgery, 187
Alaska, cold injury in, 40
Albumin, 102, 116
  in burns, 25
  intravenous, 333
  in urine, 204
Alcohol, 230
Aldosterone, 106
Alimentation, intravenous, 150
Alkali burns, of eye, 286
Alkalosis, 114
Allergy, to penicillin, 132
Alveolar-arterial gradient, 333
Alveoli, pulmonary, blast injury, 51
Ambulation—
  after vascular surgery, 220
  in spinal cord injuries, 265
Amino acid administration, 335
Amnesia, 247
Amobarbital, 149
Ampicillin, 127, 133, 287
Ampulla of Vater, 324
Amputation, 238–244
  after tourniquet use, 211
  dressings for, 242
  indications, 239
  management principles, 244
  of digits, contraindication, 353, 355
  postoperative management, 243
  rate after arterial repair, 224
  stump wound closure, 243

Amputation—Continued
technique, 239–242
traumatic, 184–185
types, 238–239
Amputation, in—
clostridial myositis, 142
cold injury, 45–46
crush injury, 205
electrical injury, 31
vascular injury, 208, 220
Amyl nitrite, 59
Amytal, 149
Analgesia, 186–195
contraindication in head injuries, 253
See also Anesthesia.
Analgesia, in—
air evacuation, 163
burns, 29
cold injury, 45
shock, 102
vascular injury, 210
Analgesics, 180
Anal stricture, 30
Anastomosis, 214–216, 331–332
duodenal, 324–325
in neck, 299
of colon, 327
of small intestine, 325
with bone fracture, 219
Anemia—
after trauma, 105
in peritonitis, 336
in renal failure, 110
Anesthesia, 186–195
conduction, 189–191
equipment, 187–188
general, 191–192
local, 189
spinal, 189
spinal and epidural, 191
techniques for, 189–195
See also Analgesia.
Anesthesia, in—
abdominal surgery, 317
blast injury of chest, 52
causalgia, 237
clostridial myositis, 141
cold injury, 41, 42
hand surgery, 354
neck surgery, 296
spinal cord surgery, 260
thoracotomy, 311–312
Anesthesiologists, 186–187
Anesthetics, 192–195
effect on hypovolemic patient, 104
for nerve block in chest wound, 310
Anesthetists, responsibilities, 186–187, 188
Aneurysm, 217
false, 213
Angiocatheter, indwelling, 286
Angiography, in arterial repair, 217, 219
Ankle:
joint fixation, 266
splinting, 231, 236
Anorexia, 74
Anoxemia, 115
Anoxia, 60, 136, 269
Antacids, 343
Antibiotics:
early administration, 179
preoperative administration in abdominal injury, 316
route of administration, 132
sensitivity studies, 198
use in air evacuation, 163
Antibiotic therapy, in—
abdominal injury, 183
abdominal surgery, 333, 334
abdominal wall defects, 345
blast injury, 52
bone and joint injuries, 227, 233
burns, 27
clostridial myositis, 140
colostomy closure, 332
ear infection, 293
ear injuries, 292
extremity injury, 184, 238
eye burns, 286
eye injuries, 284, 286, 287
head injuries, 253
infection, 119, 126–133
invasive infection, 125
local wound suppuration, 125
maxillofacial injuries, 183, 270
peritonitis, 338
pulmonary insufficiency, 116
radiation injury, 67, 78, 79
renal failure, 111
soft-tissue wounds, 197, 198, 206, 207
spinal cord injuries, 264
streptococcal myositis, 144
tetanus, 147–148, 150
vascular injuries, 222
Anticoagulants, in vascular injuries, 222
Anticonvulsants, 149–150, 253

Antidotes, for—
chemical agents, 56
glycolates, 60
Antihistamine, 33
Antiseptics, doubtful value, 197
Antitoxin:
pentavalent, for clostridial myositis, 142
tetanus, 146, 147, 148, 149
Antral balloon, 275, 279
Antrostomy, 279
Anuria, 109, 203
Apprehension:
drugs for, 189
in blast injury, 51
in clostridial myositis, 137
Arm:
anastomosis in, 216
clostridial myositis in, 133
contracture in, 223
nerve and vascular injuries, 220
preservation in injury, 238–239
splinting of, 179, 230
vessels, ligation, 218–219
See also Extremities.
Arteries:
grafts, 217
occlusion, 217
patency, 212, 223
repair, 210, 218, 238, 299–300
See also Blood vessels: Vascular injuries.
Arteriography, in—
head wounds, 253, 254
vascular injury, 212, 222
Arthrotomy, 226, 231, 232, 233
Asphyxia, 59, 157
priority of treatment, 158
tracheostomy complicated by, 302
laryngotracheal damage, 296
neck injuries, 298
Aspiration, of pericardium, 309
Atelectasis, prevention, 195
Atropine, 52, 56, 57, 189, 194, 286, 296
Auditory canal, lacerations, 292
Auricle, injuries, 291–292
Avulsion, of—
auricle, 292
blood vessels, 209
Axilla, injury, tetanus in, 145
Axillary artery, ligation, 218
Axonotmesis, 234, 235
Azotemia, in trauma, 111
Bacilli:
anaerobic spore-forming, 124
gram-negative, 122, 124, 127, 133, 137
Bacitracin, 126, 127, 133, 140
Back, wounds, debridement, 317
Bacteria
infection in wound track, 170
sensitivity testing, 126–127
See also Micro-organisms; genera of bacteria.
Bacteriology, of war wounds, 121–123
Baker intestinal tube, 335, 338
Ballistics:
behavior, 14–15
wound, 9–15
Bandages, pressure, 205
Bandaging, of sprains, 233
Barbiturates, 149, 189
Battalion aid station, 83, 179
evacuation from, 181
medical care at, 2, 165–166
sorting at, 154, 156
Battle neurosis, 50, 51
Bed rest, in kidney wounds, 348
Beta radiation, 72, 73
Bile duct, wounds, 324
Biliary system, wounds, 158, 324
Bladder:
atony, 109
care in spinal cord injury, 259, 264
ureter implantation into, 348
wounds, 348, 349
Blankets:
heating or cooling, 188
hypothermic, 163–164
Blast, from nuclear detonation, 62
Blast injury, 48–54
assisted ventilation in, 86
clinical manifestations, 50
component of multiple injuries, 82
diagnosis, 50
from nuclear detonation, 62, 66, 67–69
prevention, 54
types, 48–49
Blast injury, of—
abdomen, 52–54
chest, 51–52
ear, 50–51, 293
Blast lung, 50
Blast wave, nuclear, 67
Bleeding. *See* Blood loss; Hemorrhage.
Blister agents. *See* Vesicants.
Blistering, in crush injury, 204

Blood:
administration, 178
administration responsibility, 187
aspiration, 167
gases and pH, 188, 333
magnesium retention, 204
myoglobin absorption into, 223
nitrogen retention, 204
pathologic response to trauma, 117–118
potassium release into, 205
potassium retention, 204
type-specific, 334
urea nitrogen, 109, 111
See also Fluid.
Blood bank, 96
Blood clotting, 117, 118, 306
See also Clotting factors.
Blood count, in radiation exposure, 66, 75, 77, 78
Blood flow, 91, 193
Blood loss:
estimation, 102
in shock, 92, 93
in trauma, 104, 105
rapid, 103
See also Hemorrhage.
Blood plasma:
administration in burns, 25
loss in crush injury, 204
loss in shock, 93
replacement, 105, 205
sodium chloride concentration, 106
volume in burns, 26
Blood platelets, 117
Blood pressure:
arterial, 91, 193, 334
in crush injury, 205
in salt depletion, 108
neurological import, 249
See also Central venous pressure; Hypotension.
Blood replacement, 67, 78, 84, 206
Blood replacement, in—
chest wounds, 308
crush injury, 205
kidney wounds, 348
shock, 101
soft-tissue wounds, 197
Blood serum:
potassium, 25, 334
sodium, 107, 108
Blood study, for abdominal surgery, 316
Blood supply:
impairment, antibiotic therapy in, 126
maintenance, 196
Blood transfusion, 177
massive, 115
multiple, 117
Blood transfusion, in —
abdominal injury, 334
air evacuation, 163
peritonitis, 338
Blood type, in shock therapy, 101
Blood vessels:
erosion, 344
indirect damage, 13
intra-abdominal, injury, 318–319
major, injury, 208
See also Arteries; Capillaries; Vascular injuries; Veins.
Blood volume, 91, 300
adjustment in abdominal injury, 334
expansion, in renal failure, 11
hormonal regulation, 105, 106
in air evacuation, 163
in trauma, 104, 105
maintenance in burns, 22
restoration in shock, 97–103
restoration in trauma, 106
See also Hypovolemia.
Body surface, burned extent, 19–20
Body temperature, 188
in clostridial myositis, 137
neurological import, 249
See also Fever; Hypothermia.
Body weight, increase, 206
Bone fragments, removal in brain wounds, 251
Bone injuries, 225–233
amputation in, 239
infection, 232–244
management principles, 225–227
with vascular injuries, 219
Bone marrow, 80
Bone marrow depression, 66, 68, 69, 75, 77, 78, 79
Bony prominences, 264
Bowel. *See* Intestine: Large intestine; Small intestine.
Bowel gas, expansion, 341
Brachial artery, 214
injury, 228
ligation, 218

Bradycardia—
from head injury, 249
in shock, 93
Brain:
blood flow, 193
circulation, 191
debridement, 251
injuries, 158, 247–248
Brigade medical company, treatment at, 185
Bronchi, rupture in blast injury, 51
Bronchial tree, clearing, 307
Bronchodilators, therapy in burns, 28
Bronchoscopy, 296–297, 333
Bronchospasm, 27–28
Brooke formula, 24–26
Buck's fascia, repair, 350
Bullets:
angle, 14
See also Missiles; Rifle, bullets.
Bullet wounds, 10
See also Missile-caused wounds.
Burns, 18–34
as component of multiple injuries, 82
care in air evacuation, 163
classification, 20–21
depth of injury, 20–21
first aid, 22–23
from nuclear detonation, 62
healing delay, 79
hyperkalemia in, 194
in crush injuries, 68
infection as cause of death, 123
in nuclear combat, 65–67
management at battalion aid station, 185
muscle relaxants in, 194
of eye, 285–286, 287
pathophysiology, 22
priority of treatment, 158
water loss with, 107
See also Thermal injury.
Burr holes, 250, 254–255
Buttocks:
burns, 30
clostridial myositis in, 133
debridement of wounds of, 317
infection of wounds of, 206
tetanus in wounds of, 145
Calcium, blood level, 110
Calcium gluconate, 113, 114
Calculus, urinary, 259, 264
Caldwell-Luc operation, 275, 279
Canalization, vascular, 218
Cannulae, intravenous, 178
Canthi, inner, displacement, 278
Capillaries:
blood flow, 38, 91
permeability, 22, 38
rupture in blast injury, 51
stasis in shock, 94
Capillary perfusion, 334
Capsule, closure, 231–232
Carbenicillin, 127, 133
Carbohydrates, needs in renal failure, 112–113
Carbon dioxide tension, 33, 332
Carbon monoxide poisoning, 50, 61, 83
Cardiac arrest, 85, 111, 194, 204, 332
Cardiac output. *See* Heart, output.
Cardiac tamponade, 307, 309, 310, 311, 312
Cardiopericardial injuries, 158
Cardiorespiratory care, after abdominal surgery, 332–333
Cardiorespiratory function, maintenance, 166–177
Cardiorespiratory resuscitation, 86
Cardiovascular system, response to trauma, 104–105
Carotid artery, ligation, 219
Carotid system, external, 300
Carpal tunnel, 355
Casts:
bivalving, 219, 226, 228
care in air evacuation, 162
for soft-tissue wounds, 202
for sprains, 233
over burn, 30
plaster, 136, 228, 230–231
pressure on skin, 228
spica, 228, 230
Casts, urinary, 204
Catecholamine, level in shock, 91
Cathartics, 113
Catheter:
antibiotic perfusion by, 283
central venous, 98–100
Fogarty balloon, 212
Foley, 264, 350
for fluid administration, 112
indwelling, 26, 85, 142, 162, 183, 184, 205, 264
intravenous, 183
obstruction, 108
occlusion balloon, 320
retrograde, 325
Robinson, 350

Catheter—Continued
ureteral, 348
See also Drainage.
Causalgia, 236–237
Cavitation, 11, 17, 209, 213
Cecostomy tube, 327–328
Celiotomy, 313
Cells, death in burns, 22
Cellulitis, anaerobic, 139, 143–144, 239
Central nervous system syndrome, 74, 75–77
Central venous pressure, 98–102, 111, 115, 188, 205, 332, 334
Cephalic vein, 217
Cephaloridine, 287
Cephalothin, 127, 132, 133
Cerebral hemorrhage, 219
Cerebral injuries, 158
Cerebrospinal fluid fistula, 253
Cerebrospinal fluid leak, 163, 268, 275, 276, 278
Cerebrovascular accident, 219
Cervical spine, injuries, 262
Chemical agents, classification, 55–56
Chemical injuries, 55–61
associated injuries, 56–57, 82
burns, 19, 31–32, 285–286
management, 56–57
Chemotherapy, topical, in burns, 21, 33–34
Chest pressure-armlift artificial respiration, 172–174
Chest tube:
care in evacuation, 161–162, 310
for tension pneumothorax, 176
Chest wall, blast injury, 48
Chest wounds, 303–314
blast injury, 51–52
evacuation, 310
management in division area, 308–310
management in forward hospital, 311–313
management principles, 307–308
pathophysiology, 303–307
postoperative management, 314
respiration in, 175–177
sucking. *See* Sucking chest wounds.
See also Thorax.
Chilblain, 35, 36
Children:
external cardiac compression in, 174
fluid requirements in burns, 25
Children—Continued
mouth-to-mouth resuscitation in, 169
truncal burns in, 29
Chloramphenicol, 125, 127, 133, 140, 286, 287
Choking agents. *See* Lung damaging agents.
Cholecystectomy, 324
Cholecystitis, acalculous, 117
Cholecystostomy, 320
Choledochojejunostomy, 324
Cholinergic blockade, 57
Cholinergic stimulation, 194
Circulation:
collateral, 210, 213, 214, 218, 223
impairment in burns, 28
CIVD. *See* Cold induced vasodilatation.
Claymore mine, 10
Clearing station:
evacuation to, 156, 181
medical care at, 3, 166, 185
Clostridia:
antibiotic sensitivity, 127
proteolytic, 122
toxigenic, 121–122
Clostridial myositis, 125, 133–143
after arterial surgery, 220
as cause of death, 123
as indication for amputation, 239
clinical picture and diagnosis, 137–139
differential diagnosis, 139–140
etiology, 136
management, 141–143
pathology and bacteriology, 136–137
prophylaxis, 140–141
Clostridium:
pentavalent antitoxin, 142
species in clostridial myositis, 137, 138
species in war wounds, 122
Clostridium tetani, 122, 144, 145
Clothing, protective, 39, 46–47, 54, 58, 61
Clotting, disseminated intravascular, 117, 118
Clotting factors, 117
Cocaine, 301
Coccygectomy, 330
Cold induced vasodilatation, 37, 40
Cold injury, 35–47
as component of multiple injuries, 82
classification, 35–36, 41–43
clinical manifestations, 41–43

Cold injury—Continued
contraindication for amputation, 244
epidemiology, 38–40
first aid, 44–45
pathogenesis, 37
pathologic process, 37–38
prophylaxis, 46–47
See also Thermal injury.
Colistin, 133
Collin's spring, 161
Colloids, 102
administration, 177, 178
in burns, 25
Colon:
examination, 319
overlooked injuries, 343
wounds, 261, 325–329
Colostomy, 326, 329, 330–331, 350
closure, 331–332
contraindication, 30
retraction, 341–342
Combat type, cold injury in, 39
Communications zone:
colostomy closure in, 332
evacuation to, 207
medical support in, 3
wound closure in, 197
Compression:
cerebral, 250, 254
extravascular, 209
Compression injury, 82
Compression syndrome. *See* Crush injury.
Concussion, 257
of brain, definition, 247
of nerve, 234
Conjunctiva, sensitivity to vesicants, 58
Consciousness:
loss, 247, 254, 256
state, 248
Consultant system, 4
Contamination:
identification, 123
of colon wounds, 327
of hepatic tissue, 322
secondary, 122
Contracture:
prevention, 236, 243, 355
Volkmann's ischemic, 223
Contusion, of—
blood vessels, 209
brain, definition, 247–248
eye, 287
Contusion, of—Continued
eyelid and eyeball, 284
nerve, 235
Convulsions, 57, 77, 110
drugs for control, 149
in tetanus, 145, 150
Copper sulfate, 32, 285
Cornea:
abrasion, 283, 284
foreign bodies on, 287
laceration, 283, 289–290
nerve agent absorption, 57
sensitivity to vesicants, 58
Cortisone, administration in trauma, 106
Costal cartilage, fracture, 307
Cough, 58
improvement in chest wounds, 310
ineffective, 51
laryngeal, 296
Cranial tongs, care in air evacuation, 161
Craniectomy, 254
Craniocerebral wounds and injuries, 181–182, 247–256
classification, 247–248
evaluation, 248–249
expedient measures, 256
management, 249–255
prognosis, 256
See also Head wounds.
Creatinine, leakage in crush injury, 203
Cricothyroidotomy, in extreme emergency, 302
Crush injury, 68–69, 203–206
as component of multiple injuries, 82
associated with blast injury, 50
clinical considerations, 203–204
management, 205–206
of chest, 176
of spinal cord, 257
pathogenesis, 203
Crutchfield calipers, 262
Crystalloids, 102
administration, 177, 178
after trauma, 105
Culture technique, 198
Curare, 192, 194, 195
Curarelike drugs, therapy of tetanus, 150
Curarization, 116
Cyanides, 56
Cyanmethemoglobin, 59
Cyanogen agents, 58–59

Cyanogen chloride, 58, 59
Cyanosis, in—
  blast injury, 51
  cold injury, 41, 42
  pneumothorax, 304
  shock, 94
  vascular injury, 210
Cyclopentolate hydrochloride, 284
Cycloplegia, 284
Cyclopropane, 193
Cystic duct, wounds, 324
Cystography, 349
Cystostomy, suprapubic, 184, 264, 349, 350
Deafness, 293
  See also Hearing loss.
Debridement:
  anesthesia for, 189
  principle, 17, 123
  See also Surgery.
Debridement, in—
  abdominal wall wounds, 331
  auricle wounds, 291–292
  back and buttocks wounds, 317
  bone and joint injuries, 225, 226, 227, 232, 233
  burns, 30, 33
  chest wounds, 311, 313
  clostridial myositis, 140, 141
  cold injury, 45
  duodenal wounds, 324
  electric injury, 31
  external genitalia wounds, 350
  extremity injuries, 238
  eye wounds, 287
  genitourinary tract wounds, 347
  hand wounds, 354–356
  head wounds, 250–253, 254, 256
  kidney wounds, 348
  laryngotracheal injuries, 297
  liver wounds, 319, 320, 322
  maxillary sinus wounds, 278–279
  maxillofacial wounds, 267, 270
  nasal wounds, 276
  nerve injury, 235–236
  renal failure, 111, 112
  soft-tissue wounds, 196–197, 198–202
  spinal cord wounds, 260
  stomach wounds, 324
  tetanus, 146
  vascular injuries, 212–213
Deceleration injury, 82
Decompression:
  gastrointestinal, 263
  of spinal cord, 260–261
Decompression injury, 82
Decongestants, in—
  aero-otitis, 294
  inner ear injury, 294
Decontamination, 56, 57, 65
Decubitus ulcers:
  from cast pressure, 229
  prevention, 182, 259, 264–265
  See also Necrosis, pressure; Skin, breakdown.
Defibrillation, 188
Dehiscence, wound, 331, 341
Dehydration—
  at aid station, 180
  in trauma, 107
  of tracheobronchial tree, 115
Delirium, 60
Demerol, in burns, 23
Density, of tissues, 14
Dental laboratories, 267
Dentures, 271–272
Dexamethasone sodium phosphate, 286
Dextran, 102
Dextrose, in—
  burns, 25
  trauma, 101, 107
Diabetes, 121
Dialysis, renal. *See* Renal dialysis.
Diaphragm:
  elevation, 263, 337
  hernia, 309
  injury, 322
Diarrhea, 74, 93
  bloody, 77
  in renal failure, 110
  salt depletion with, 108
Diet, in—
  cold injury, 45
  maxillofacial wounds, 273
  spinal cord injury, 259
Digitalis, 101
Digital nerves, neurorrhaphy, 235
Digits:
  amputation contraindicated, 353, 355
  exercise, 236
Digoxin, 334
Diplopia, 268, 274, 276
Dish-face deformity, 276
Dislocations, 158, 223
  in hand, 356
  of cervical spine, traction for, 262

Dislocations—Continued
of spinal cord, 257
reduction, 233, 239
Diuresis, in—
crush injury, 204
renal failure, 113
Diuretics, 101, 116, 333
osmotic, 31, 253, 254
Diuretics, in—
blast injury, 52
burns, 26–27
crush injury, 205
Division area:
chest wound management in, 308–310
eye injury care in, 285–286
hand wound care in, 353–354
nerve injury care in, 236
primary treatment in, 165–185
sorting in, 154
Drainage:
inadequate, 120
intercostal tube, 308, 312, 313
kidney, 265
of sinuses, 277–278
retropubic, in bladder wounds, 348, 349
T-tube, 320, 324, 348
urinary, 251, 264
See also Catheter.
Drainage, in—
abdominal abscesses, 344–345
abdominal wounds, 334
abscesses, 338
colon wounds, 327, 329
duodenum wounds, 325
genitourinary tract wounds, 347
hip joint wound, 232
liver wounds, 319, 320, 322
pancreatic wounds, 323, 323–324
pharynx and esophagus wounds, 298
rectal wounds, 330
soft-tissue wounds, 202, 206
ureteral injuries, 348
urethral injuries, 349–350
Dressings, 179
changes, 164
nonocclusive, in bone and joint injuries, 226, 227
pressure, 177, 211, 273
Velpeau, 228
Dressings, for—
amputations, 242
burns, 23, 30, 32
chest wounds, 310
Dressings, for—Continued
cold injury, 45
evisceration, 183
extremity wounds, 184–185
hand wounds, 354, 356–357
head wounds, 256
joint injuries, 232
maxillofacial wounds, 267, 271
soft-tissue wounds, 197, 202
Droperidol, 194
Drugs. *See* Medications; specific types of drugs.
Duodenectomy, 324
Duodenum:
examination, 319
pathologic response to trauma, 116–117
wounds, 324–325
Dura:
debridement, 251
examination, 254
sinuses, 255
wound closure, 251, 260
Dysphagia, 296
Dysphonia, 296
Dyspnea, in—
blast injury, 50, 51
hemothorax, 306
larynx or trachea damage, 296
pneumothorax, 304
Ear:
blast injury, 50–51
wounds and injuries, 291–294
Eardrum. *See* Tympanic membrane.
Earplugs, 54
Ecchymoses, 78
Echelons, of medical care, 2–4
Edema, 136, 257, 258, 268, 271, 277
after arterial repair, 220
blocking airway, 296
causing respiratory emergency, 167
cerebral, 253
fasciotomy for, 223
pulmonary, 52, 59, 60, 61, 100, 101, 110, 115, 204, 314
See also Swelling.
Edema, in—
burns, 22, 28
clostridial myositis, 138
cold injury, 41, 42, 43
Elbow:
joint, splinting, 230
preservation, 238

Electrical injury:
as component of multiple injuries, 82
burn therapy in, 31
diuretic use in, 26
Electric current, burns from, 19
Electrocardiography, 188
characteristics of, in potassium intoxication, 110
in crush injury, 205–206
Electrocautery, in head wounds, 251, 254
Electrolytes, 23
administration responsibility of, 187
balance of, after head surgery, 251
management of, in peritonitis, 338
physiologic response of, to trauma, 106–108
Electrolytes, in—
burns, 24, 25
renal failure, 111
shock, 101
Elevation, of—
cold injured part, 45
limb, 197, 206, 228, 239–240
Embolism, air, 298
Emphysema:
of face and neck, 268, 296
subcutaneous, 304, 306
subcutaneous and deep-tissue, 140
tracheostomy complicated by, 302
Endoscopy, 297
Endotracheal equipment, 188
Endotracheal intubation, 52, 114, 115, 188, 297, 308, 309, 333
Endotracheal intubation, in—
air evacuation, 161
head wounds, 249
respiratory emergency, 167
Enema, 251, 263
Energy, kinetic. *See* Kinetic energy.
Enucleation, of eye, 288–289
Environmental factors, in cold injury, 38–39
Enzymes, bacterial, in war wounds, 122–123
Epilepsy, 21
Epinephrine:
contraindication in hand surgery, 354
in trauma, 106
Equipment, triage and resuscitation facility, 96–97
Erythema, in crush injury, 204
Erythrocytes, concentration in trauma, 105
Erythromycin, 127, 133, 140
Escharotomy, in burns, 28–29
Eschars, 42, 45
Esophagus:
perforations, repair, 313
wounds, 297–298
Ethacrynic acid, 27, 113
Ether, 192–193
Ethmoidal bone, 268
Ethmoidal sinuses, fractures, 278
Ethmoidectomy, 278
Eustacian tube, 293
Evacuation, 3, 154–155, 181
in Vietnam war, 156
secondary, 154
See also Air evacuation; Helicopter evacuation; Patients, movement.
Evacuation, in—
abdominal wounds and injuries, 315–316, 335, 339, 340, 341
amputation, 243, 244
blast injury, 52
bone and joint injuries, 226
burns, 34, 65
cervical spine injuries, 262
chest wounds, 310
craniocerebral injuries, 182
crush injuries, 206
eye wounds, 290
fractures, 228
genitourinary tract wounds, 184
hand wounds, 353
head wounds, 253, 256
inner ear injuries, 294
maxillofacial injuries, 183, 270, 279–280
multiple injuries, 83–84
nuclear injuries, 63–64
oliguria, 113
radiation injuries, 81
shock, 178
soft-tissue wounds, 206–207
spinal cord injuries, 259
thoracoabdominal wounds, 310
vascular injuries, 208
Evisceration, 183
Excision, inadequate, 120
Excision, in—
clostridial myositis, 140, 142
invasive infection, 125
tetanus, 148–149
wound debridement. *See* Debridement.
Exercise:
after amputation, 248

Exercise—Continued
deep breathing, 266
of digits, 236
of joints, 231
passive, 266
Explosives, burns from, 19
Exteriorization, of—
bowel, 341
colon wounds, 326, 328–329
intestine, 318
External cardiac compression, 174–175
External ear, wound and injuries, 291–293
Extremities:
characteristics in vascular injury, 210
elevation, 206, 228
exercise for, 266
postoperative position, 220
preservation, 238
surgery of, 197
wounds and injuries, 184–185, 191, 198
See also Arm; Leg.
Eye:
examination and diagnosis, 282–283
level, 268
movement, neurological import, 248
muscle imbalances, 278
removal, 288–289
wounds and injuries, 158, 281–290
Eyelid:
laceration, 287
spasm, 58
Face:
decontamination, 56
emphysema, 296
injuries, 172
wound closure, 202
wound infection, 206
wound repair, 267
Facial bones, fractures, 274–277
Facial nerves:
neurorrhaphy, 235
palsy, 294
Fascia lata, 251, 260
Fascial decompression, 239
Fasciitis, 143–144
Fasciotomy, 28, 202, 226
in crush injury, 205
in electrical injury, 31
with arterial repair, 220–222, 223
Fat:
embolization, 229
needs in renal failure, 113
Fatigue, role in cold injury, 39–40
Feces:
bloody, 54
impaction, 264
Femoral artery, 214, 218
Femoral vein, 217–218
Femur:
amputation through, 243
splinting, 85, 230–231
Fever, postoperative, 337
Fibrinogen, 118
Fibula, fracture, 120
Field medical system, in nuclear warfare, 63
"Fight or flight," 106
Fire-rescue personnel, 19
First aid:
in first echelon, 2
See also Resuscitation.
First aid, in—
bone and joint injuries, 226
burns, 22–23
chest wounds, 308–310
cold injury, 44–45
eye injuries, 285–286
multiple injuries, 84
Fission products, 70, 72
Fistula:
abdominal, 346
arteriovenous, 210, 213
biliary, 322
biliary-pleural, 322
cerebrospinal fluid, 253
enteric, 93
formation in maxillofacial wounds, 267
gastrointestinal, 108
intestinal, 345
mucous, 326–327, 329, 330–331
tracheoesophageal, 302
Fixation:
in malar-maxillary complex, 275
intermaxillary, 271, 272, 273, 279
of mandibular fractures, 274
of palate, 277
Fixature externe, 219, 228
Flail chest, 116, 306–307, 309
after crush injury, 176–177
airway control in, 308
Fluid:
administration route, 112, 180, 182
intake in renal failure, 111–112
management in peritonitis, 338
needs in burns, 24–26

Fluid—Continued
overload, 114
postoperative care, 195
retention in crush injury, 204
volume loss in shock, 92, 93
See also Blood; Colloids; Crystalloids; Electrolytes, specific fluids.
Fluid administration, in—
abdominal surgery, 334, 335
burns, 22, 23, 34, 185
chest wounds, 314
clostridial myositis, 142
crush injury, 205
hemorrhage, 177
kidney wounds, 348
multiple injuries, 84, 86
pulmonary insufficiency, 115
renal failure, 111
shock, 97–103, 178–179
soft-tissue wounds, 197, 206
spinal cord injuries, 263, 264
Fluid balance:
after head surgery, 251
in maxillofacial wounds, 267, 268
in spinal cord injury, 259
maintenance, 114
Fluorescein strip, 284
Fluoroscopic examination, 337
Fluothane, 52
Fogarty balloon catheter, 212
Foley catheter, 264, 350
Foot:
paresthesia, 41
splinting, 231
Footgear, for cold protection, 46–47
Foot supports, 266
Foreign bodies:
aspiration, 167
identification, 197
removal, 140, 196, 199, 200, 201, 206
retention in wound, 120
Foreign bodies, in—
arterial injury, 217
chest, 311
eye, 285, 287
larynx, 167
Foreign body removal, in—
bone and joint injuries, 225–226, 227, 233
eye injury, 282, 283
hand wounds, 355
head wounds, 250, 251, 256
joint injury, 231
maxillofacial wounds, 269
Foreign body removal, in—Continued
pleura injury, 312
spinal cord injuries, 260
Forward area:
amputation management in, 238, 244
anesthesia equipment, 188
Forward hospitals:
bone and joint injury care at, 227, 232
care at, 166
causalgia in, 236
chest wound management in, 311–313
eye injury care at, 286–290
multiple injury care at, 85
neurosurgical care at, 247
wound closure in, 197
Forward surgery, 1–5
Foster frame, 260, 262
Fractures, 227–231
associated with arterial injuries, 219
associated with burns, 30
external fixation, 219, 228
facial, 273–278
healing delay, 69, 79
maxillofacial, 268, 271–272
priority of treatment, 158
reduction, 227–228, 239
splinting, 22, 85, 179
thoracic, 307
Fractures, of—
cervical spine, 262
ear bones, 292
facial bones, 274–277
hand bones, 356
laminae, 260
paranasal sinuses, 277–279
pelvis, 350
skull, 254
Frontal sinuses, fractures, 277–278
Frostbite, 35, 36, 37, 39
Fuels, source of burns, 18, 19
Furosemide, 27, 113, 205, 333
Gagging facies, 268
Galea, 251
Gallbladder:
pathologic response to trauma, 117
wounds, 324
Gamma radiation, 69, 70, 72, 73, 80
Ganglionic blockage, 95
Gangrene:
after ligation, 218
amputation indicated for, 239
anoxic, 140
gas. *See* Clostridial myositis.

Gangrene—Continued
in cold injury, 43
See also Necrosis.
Gas:
bowel, expansion, 341
in anaerobic cellulitis, 139, 143, 144
in subphrenic abscess, 337
production in clostridial myositis, 138
Gas abscess, 124
Gastrectomy, 324, 343
Gastric cooling, 343
Gastrografin, 344
Gastrointestinal decompression, 335
Gastrointestinal syndrome, 74, 75, 77–78
Gastrointestinal tract, electrolyte loss, 106
Gastrostomy, 313, 335
Gelfoam, 256
Genitalia, external, wounds and injuries, 184, 350–351
Genitourinary tract:
examination, 319
wounds and injuries, 158, 184, 316, 347
Gentamicin, 127, 133, 286
Gibbus, 258
Glucose, 113, 114
administration, 335
administration in renal failure, 113
Glycogen, mobilization, 92, 106
Glycolates, as incapacitants, 60–61
Grafts:
autogenous vein, 299
autologous artery, 217
autologous vein, 214, 217, 218
homologous artery-bank, 217
in maxillofacial wounds, 267, 271
skin, 46, 232, 243, 251, 345
vascular, 217
Guérin fractures, 277
Guncrews, protection from blast injury, 54
Hair, loss after radiation exposure, 75
Hallucinations, 60
Halothane, 141, 193, 194
Hand:
importance of preservation of, 238
splinting, 230, 236
wound closure, 356–357
wounds and injuries, 184, 352–357
Headache, 254
Head wounds, 93, 181–182
closed, 254–256
closure, 202
Head wounds—Continued
open, 250–253
priorities in treatment, 249
tangential, 253–254
See also Craniocerebral wounds and injuries
Healing:
delay, 69, 79
promotion, 196, 202, 218, 233
Hearing loss, 51, 294
See also Deafness.
Heart:
arrhythmia, 32
external cardiac compression, 174–175
output, 22, 91, 105, 191, 193
sounds, 188
wounds, 307, 311, 312–313
See also Cardiac arrest.
Heart failure, congestive, 110
Heimlich chest drain valve, 162, 176, 309
Helicopter evacuation, 63, 84, 154, 155, 156, 159, 165, 181, 208, 290
See also Air evacuation; Evacuation.
Hematocrit, in—
abdominal injury, 316
air evacuation, 163
crush injury, 204
shock, 102
trauma, 105
Hematoma, 120, 268, 271
in blast injury, 51–52
infected, 206
in head wounds, 251
in neck, 298–299
intracerebral, 256
pericardial, 307
pulsating, 210, 218
retroperitoneal, 348
subdural or intracerebral, 254
subintimal, 213
Hematoma, of—
auricle, 292
blood vessels, 209, 210
intestine, 325
scalp, 254
subdural, 255
Hematopoietic syndrome, 74, 75, 78
Hematuria, 53, 85, 348
Hemiplegia, 254
arterial ligation in, 219
hyperkalemia in, 194
Hemochromogen, 26, 31
Hemoptysis, 51, 296

Hemorrhage, 136, 157, 257, 258
abdominal, 183, 315, 316–317, 318–319
after arterial repair, 222–223
blocking airway, 296
cardiac, 313
cerebral, 219
concealed, 94, 103
extradural, 255
gastrointestinal, 116–117
intra-abdominal, 183, 344
intracranial, 254
intrapleural, 312
intrathoracic, 311
ocular, 283, 284
of liver wounds, 320, 322
of rectal wounds, 329
of tympanic membrane, 293
postoperative control, 195
priority of treatment, 158
secondary, amputation indicated for, 239
stress ulcer, 343
subadventitial, 213
subconjunctival, 284
suppression, 213
tracheostomy complicated by, 302
See also Blood loss.
Hemorrhage, in—
duodenal injury, 324
maxillofacial wounds, 267, 268
nasomaxillary injury, 275
neck injuries, 298
radiation sickness, 78
vascular injury, 210
Hemorrhage control, 56, 84, 106, 166, 177–178, 183, 184
See also Hemostasis.
Hemorrhage control, in—
burns, 18, 22, 23
external genitalia injuries, 350
genitourinary tract wounds, 347
hand wounds, 353
injured extremities, 238
liver wounds, 322
maxillofacial wounds, 269
shock, 97
vascular injury, 211–212
Hemostasis, 201
in hand surgery, 354
in neck injuries, 298
of liver wounds, 319, 320
See also Hemorrhage control.
Hemostat, for hemorrhage control, 177
Hemothorax, 305–306
Hemotympanum, 50
Heparin, 118, 230
Hepatectomy, 320
Hepatic duct, wounds, 324
Hepatic veins, hemorrhage, 320
Hernia:
cerebral, 250
diaphragmatic, 309
Hip, contracture prevention, 243
Hip joint:
injury, 232
splinting, 230–231
Homatropine hydrobromide, 284
Hormones:
adrenal, after trauma, 105–106
antidiuretic, 106
pituitary, after trauma, 105–106
secretion in shock, 92
Hospitals:
evacuation, sorting at, 154
evacuation to, 156, 181
field, 64, 154
forward. *See* Forward hospitals.
general, medical care at, 3
medical care at, 3
Humerus:
fracture, 228
splinting, 85, 230
Humidification, during air evacuation, 160
Hydration, 26, 180
Hydrocortisone, secretion in trauma, 106
Hydrogen cyanide, 58, 59, 61
Hydrogen peroxide, 149
Hydrostatic dilatation, 217, 223
Hyoid bone, injuries, 269
Hyperalimentation, 107, 335
Hyperemia, in cold injury, 41, 42
Hyperhidrosis, in cold injury, 41, 42, 46
Hyperimmune globulin, human tetanus, 147, 148
Hyperkalemia, 111, 194
in crush injury, 204
therapy, 113
See also Blood, potassium; Potassium.
Hyperphosphatemia, 32
Hyper-Tet, 147, 148
Hyphema, 283, 284
Hypocalcemia, 32
Hypocapnea, 115, 116
Hypocoagulability, in trauma, 117
Hypogastric artery, 217

Hyponatremia:
in renal failure, 110
See also Salt; Sodium.
Hypoproteinemia, 110
Hypotension, 249
anesthesia in, 193
curare use causing, 195
See also Blood pressure.
Hypotension, in—
crush injury, 203, 204, 205
shock, 92, 93, 94
trauma, 104, 107
Hypothermia, gastric, 117
Hypothermic blankets. *See* Blankets, hypothermic.
Hypovolemia:
anesthesia in, 191, 193, 194
in trauma, 104, 105
muscle relaxants in, 194, 195
See also Blood volume.
Hypoxemia, 27, 116, 249, 314, 332
Hypoxia, postoperative, 195
Ileocolostomy, 327–329
Ileostomy, 326–327
retraction, 341
Ileus, 93, 335
management in burns, 29
paralytic, in spinal cord injuries, 262, 263
postinjury, 344
postoperative, 341
Immersion foot, 35, 36, 37
Immobilization:
external, for fractures, 228
inadequate, 121
in position of function, 230
See also Splinting.
Immobilization, of—
bone and joint injuries, 226
cervical spine injuries, 262
fractures, 228
hand, 353, 354
injured extremities, 238
jaw, 271–272
joint injuries, 232
knee, 230, 231
soft-tissue wounds, 197, 202
spinal cord injuries, 259
Immunization:
active, against tetanus, 146
against tetanus, 146
combined active and passive, 147
passive, against tetanus, 146–147
Incapacitants, 60–61
Incision:
circumferential, in amputation, 240
escharotomy, 28, 29
for tracheostomy, 300–301
in bone and joint injuries, 225–226, 227
in soft-tissue wound debridement, 198–199
Kraske, 330
operative, 342
Incubation period, 119
of tetanus, 144–145
Infants, mouth-to-mouth resuscitation in, 169
Infection, 119–150
after arterial repair, 218, 220, 222–223
after prosthetic use, 217
after splenectomy, 322
amputation indicated for, 239
antibiotic therapy. *See* Antibiotic therapy; specific names of antibiotics.
classification, 123–125
control, 179
decubitus ulcers, 264
etiology, 120–121
invasive, 123, 124, 125
of skull, 250
of tympanic membrane, 293
prevention in bone and joint injury, 225, 227
prevention in maxillofacial wounds, 269–270
prevention in spinal cord injuries, 264
urinary, 259
Infection, in—
bone and joint injuries, 228, 232–233
dressing change, 164
injured extremities, 238
internal fracture fixation, 219
joint injury, 231
liver wounds, 322
maxillofacial wounds, 267, 268
nerve wounds, 235
pulmonary insufficiency, 114, 115
radiation injury, 67, 78, 79
renal failure, 110
septic shock, 94, 95
soft-tissue wounds, 206
spinal cord injuries, 260
wound track, 17
Infraorbital nerve, 274

Inhalation injury, 61
edema after, 167
See also Smoke inhalation.
Inner ear, wounds and injuries, 294
Inotropic agents, 101
Instruments, for hand surgery, 354
Insulin, 113, 114
Interorbital space, 278
Intestinal obstruction, 93, 344, 345
Intestinal tract, examination, 319
Intestine:
exteriorization, 318
fluid loss in shock, 93
function, care in spinal cord injuries, 262–263
large. *See* Large intestine.
small. *See* Small intestine.
Intima, injury, 213
Intraocular contents, prolapse, 283
Iodine compounds, radiopaque, 344
Iris, damage, 283
Irradiation:
partial body, 80
whole body, 73–79
Irrigation, of—
abdominal abscesses, 345
arterial lumen, 216
bone and joint wounds, 226, 227, 233
brain wounds, 251
chemical burns of eye, 285–286
colon, 332
eye, 282, 284, 286, 287
hand wounds, 355
head wounds, 256
joint injury, 231
rectal wounds, 331
sinus, 278–279
urinary tract, 264
wounds, 199, 201
Irritants, 58
Ischemia, 103, 120, 211, 222, 223, 235
Isoproterenol, 101, 334
Jaw:
abnormality, 273
immobilization, 271–272
spasm, 169
Jejunostomy, 313, 335, 338
Jejunostomy tube, 344
Jejunum, 325
Joint capsule, wound closure, 202
Joints:
arterial repair at, 216
immobilization, 228
Joints—Continued
injuries, 225–233
sprains and dislocations, 233
Jugular systems, internal, 300
Kanamycin, 133
Kayexalate, 113
Ketamine, 141
Kidney:
drainage, 265
pigment deposition in, 203
responses to trauma, 108–114
wounds, 261, 347–348
See also Renal insufficiency; Uremia.
Kinetic energy:
formula, 9–10
of missile, 11, 13, 14, 15
Kirschner wires, 228, 356
Klebsiella, species in war wounds, 122
Knee:
flexion deformity, 266
preservation, 238
splinting, 230–231
Korean war:
amputation rate and arterial repair, 224
clostridial myositis in, 140
cold injury in, 35, 40
missile wounds, 13, 16
sorting, 154
Kraske incision, 330
Kyphosis, 258
Laceration, of—
arteries, 212, 214
blood vessels, 209
brain, definition, 248
cerebral arteries and sinuses, 255
ear, 291, 292
eye, 283, 289–290
eyelid, 287
nerve, 235
Lacrimination, 58, 59
Lactic acid, leakage in crush injury, 203
Laminae, fractures, 260
Laminectomy, 258, 259–260
Laparotomy, 315, 317–332
in intra-abdominal injury, 54
repeated, 331
See also Abdomen.
Large intestine, blast injury, 54
Laryngeal spasm, 269
Laryngoscopy, 296–297

Larynx:
edema, 167
foreign bodies in, 167
wounds, 296–297
Latent period, in radiation sickness, 75
Lavage, of chemical burns, 31–32
Leg:
clostridial myositis in, 133
contracture in, 233
injury, tetanus in, 145
splinting, 179, 231
vessels, ligation, 218–219
See also Extremities.
Leukocytes, in peritonitis, 336
Leukocytosis, in renal failure, 110
Leukopenia, 67
Lidocaine, 189, 191
Ligation, 240
amputation rate after, 224
in infected arterial repair, 223
of concomitant vein, 217–218
of neck arteries, 300
vascular, 218–219
Limbs. *See* Arm; Extremities; Leg.
Lips, protection, 273
Litter, double, preparation, 265
Liver:
displacement, 337
wounds, 86, 319–332
Lobectomy, 320
Lumbar fascia, 261
Lumbar puncture, 258
Lumbar venous plexus, 344
Lumen, arterial, 214–216
Lung:
blast injury, 48, 51–52
collapse, 304
compression, 304
pathologic responses to trauma, 114–116
resection contraindicated, 312
tissue density, 14
water loss from, 107
Lung damaging agents, 55, 59–60
Lymph system, 125
Mafenide, 33, 292
Magnesium:
retention in blood, 204
toxicity, 113
Malar bones, 276
Malar-maxillary complex, fractures, 274–275
Malocclusion, 267, 276, 277
See also Teeth.
Mandible:
damage, 268
fractures, 273–274
Mandibular stump, 271
Mannitol, in—
burns, 26
crush injury, 205
renal failure, 111, 113
Marlex mesh, 331, 345
Marsupialization, for urethral injuries, 349
Mask, protective. *See* Protective mask.
Mass casualties, 4–5
blast injuries, 67–69
burns, 18
cold injury, 43
crush injuries, 68–69
in thermonuclear warfare, 62–81
radiation injuries, 69–81
thermal burns, 65–67
translational injuries, 69
Masseter muscle, spasm, 145
Materiel lists, field medical, 165
Maxilla, fracture, 268
Maxillary sinuses, 278–279
Maxillofacial wounds, 183, 267–280
diagnosis, 268
evacuation, 279–280
fractures, 271–272, 273–278
initial surgery, 270–271
management, 267, 268–270
postoperative management, 273
priority of treatment, 158
transportation, 181
Median nerve, 355
Mediastinum:
displacement, 304, 306, 307
injury, 311
Medical attendants, in air evacuation, 164
Medical facilities, in combat, 154
Medical officers, responsibilities, 154–155
Medical service, mission, 153, 157
Medications:
in cold injury epidemiology, 40
preoperative, 189
use in air evacuation, 163
See also specific types of drugs.
Medicine, military. *See* Military medicine.
Melena, in blast injury, 53
Meningeal artery, 255
Meninges, infection, 276

Meningitis, 278
  gram-negative bacillary, antibiotic therapy, 133
  infection as cause of death, 123
Mental confusion, in trauma, 107
Meperidine, 189
Methemoglobin, 59
Methicillin, 127, 133, 287
Micro-organisms:
  classifications, 121–122
  invasive, 121–122
  local, 122
  See also Bacteria; genera of micro-organisms.
Micturition, difficulty, 145
Middle ear, wounds and injuries, 293
Military discipline:
  breakdown, 60
  in cold injury, 40
Military medicine:
  role, 2
  standards, 165
  success, 1
  teamwork concept, 1
Military surgery, principles, 1–5, 154
Missile-caused wounds, 9–17, 82
  distribution, 16–17
  exit and entrance, 15
Missiles:
  ballistic behavior, 14–15
  characteristics, 10
  high-velocity, 209, 212, 213, 214, 217, 254, 257, 320
  low-velocity, 319
  mass and size, 10
  secondary, 68
  velocity, 10–13
Mobility—
  in echelons, 4
  of patients, 159–160
Moisture, role in cold injury, 38
Mood changes, 60
Morale disturbance, incapacitants in, 60
Morbidity:
  decreased, 159
  in radiation injury, 67, 68
  of rectal wounds, 329
  of urethral injuries, 349
  prolonged, 199
Morphine, 23, 54, 92, 102, 182, 189, 296
Mortality:
  decreased, 159
  See also Survival rate.
Mortality, in—
  abdominal surgery, 339
  abdominal vessel injury, 318
  abdominal wounds, 315
  colon wounds, 326
  crush injury, 204
  duodenal wounds, 324
  liver wounds, 320
  neck injuries, 298
  postoperative peritonitis, 335
  radiation injury, 67, 68
  rectal wounds, 329
  septicemia, 338
Motor examination, 248–249
Mouth:
  floor of, edema, 167
  hygiene, 270
  infection, 269
  injury, 169
Mouth-to-mouth artificial respiration, 166, 168–169
Mouth-to-mouth via artificial mouth airway respiration, 172
Mouth-to-nose artificial respiration, 169–172
Mucolytic agents, therapy in burns, 28
Mucosa, oral, 271
Mucous surfaces, 57
Multiple injuries, 82–87
  abdominal, 317–318
  etiology, 82–83
  management, 83–87
  maxillofacial, 267
  nerve injuries in, 234
  priority of treatment, 158
Multiple injuries, in—
  blast injury, 50
  chemical injury, 56–57
  cold injury, 40
  duodenal wounds, 324
  nuclear blast, 64, 68
  radiation sickness, 78–79, 80–81
  rectal wounds, 329–330
  spinal cord injury, 261
  translational injuries, 69
  vascular injuries, 209
Multiple injuries, of—
  chest, 303
  colon, 329
  extremities, amputation in, 239

Multiple injuries, of—Continued
head and neck, 270
neck, 295, 298
Muscle:
changes in clostridial myositis, 139
characteristics in crush injury, 204
characteristics in soft-tissue wounds, 199
damage, 198, 239
exercises, postoperative, 220
for covering arterial repair, 218
irritability, 145
massive damage, 158
spasm, 144, 145
tissue density, 14
Muscle relaxants, 194
with droperidol, 194
with nitrous oxide, 194
Musculofascial tissue, 218
Mustard gas, 58
Myelography, in spinal cord injury, 258
Myoglobin, absorption into blood, 223
Myositis:
amputation contraindicated in, 239
anaerobic streptococcal, 139
clostridial. *See* Clostridial myositis.
streptococcal, 144
Myringotomy, 294
Narcotics, 180
contraindication, 253, 259, 268
postoperative, 195
Narcotics, in—
air evacuation, 163
burns, 185
flail chest, 177
pain control of chest wounds, 310
shock, 102
Nasofrontal duct, 277
Nasogastric decompression, 341
Nasogastric intubation, 29, 34, 162, 167, 169, 183, 251, 263, 298
Nasogastric suction, 335
Nasomaxillary complex, fractures, 275–276, 276–277
Nasotracheal aspiration, 27
Nebulizer, use in air evacuation, 161
Neck:
emphysema, 296
wounds and injuries, 295–302
Necrosis, 223
acute tubular, 109
byproducts, 223
coagulation, 22, 31
of colostomy or ileostomy, 341
Necrosis—Continued
of wound edges, 345
pressure, 264–265
skin, 236
tissue, 111, 136
See also Decubitus ulcers; Gangrene.
Negro, susceptibility to cold injury, 37, 40
Neomycin, 126, 127, 133, 233, 286, 345
Neostigmine, 263
Nephrectomy, in kidney wounds, 348
Nephrosis, lower nephron, 109
Nephrostomy, 348
Nerve agents, 56, 57
Nerve block, 191
Nerve block, in—
chest wounds, 310
hand surgery, 354
Nerve roots, injury, 260
Nerves:
injuries, 220, 239, 300
intercostal, injury, 51
of hand, 355
peripheral. *See* Peripheral nerves.
repair, contraindication, 227
transection in amputation, 242
Nervous system:
autonomic, in shock, 93
central, 92
central, syndrome. *See* Central nervous system syndrome.
Neurapraxia, 234
Neurogenic shock, 92, 93, 94, 103
Neurologic evaluation, 248–249
Neurologic examination, 258
Neurorrhaphy:
in hand, 356
in neck, 300
primary, 235
Neurosurgical teams, 247
Neurotmesis, 234, 235
Neutron radiation, 69, 70, 72, 80
Nitrite, 56, 59
Nitrogen, retention in blood, 204
Nitrous oxide, 52, 141, 194
Noradrenalin. *See* Norepinephrine.
Norepinephrine, 103, 106
Nose, 275–276
blowing contraindicated, 278, 293, 294
infection, 269
running, 57
Nuclear reactions, 70
Nuclear weapons, 62–63, 65
See also Thermonuclear warfare.

Nurse anesthetists, 186–187
Nursing care—
  for prevention of decubitus ulcers, 264–265
  in tetanus, 150
Nutation, 14
Nutrition, in spinal cord injury, 259
Nylon, as wound covering, 345
Occlusion, of blood vessels, 209
Odor:
  bitter almond, 59
  in clostridial myositis, 138
  new-mown hay, 59
Oligemic shock, 92, 93–94, 103
  in crush injury, 203, 204
Oliguria, 109
  evacuation in, 113
  in renal failure, 111
  in tetanus, 145
  in trauma, 111
  management in burns, 26–27
Ophthaine, 284
Ophthalmia, sympathetic, 288
Ophthalmoscope, in sweat gland examination, 234
Ophthetic, 284
Opiates, contraindication, 237
Opisthotonos, in tetanus, 145
Oropharyngeal intubation, 167, 172
Ossicular chain, 50
Osteomyelitis, pelvic, 330
Otitis:
  aero-, 293–294
  external, 292–293
Otorrhea, 268
Oxacillin, 127, 133
Oxime, 57
Oxygen:
  administration, 187, 338
  equipment for therapy, 188
  high concentration, 115
  postoperative administration, 195
Oxygenation, hyperbaric, 142–143, 144
Oxygen tension, 116, 163, 332
Oxygen therapy, 59, 60, 61, 300
Oxygen therapy, in—
  blast injury, 52
  chest wounds, 314
  fracture cases, 229
  smoke inhalation, 27
Packing, of sinus, 278, 279
Pain, 138
  relief, 23, 180, 184, 195, 310
Pain, in—
  abdominal abscess, 337
  arterial repair, 220
  blast injury to abdomen, 53, 54
  clostridial myositis, 137
  cold injury, 42
  nerve injury, 236
Palate:
  fracture, 277
  swelling, 269
Palsies, 235–236, 294
Pancreas, wounds, 322–324
Pancreaticoduodenectomy, 324–325
Pancreatic system, wounds, 158
Papavarine hydrochloride, 228
Paradoxical respiration. *See* Respiration, paradoxical.
Paraldehyde, therapy of tetanus, 149
Paralysis:
  ileus, in spinal cord injury, 262
  in crush injury, 204
  in spinal cord injury, 257, 258, 259
  muscle use in, 265
  of bladder, 264
Paranasal sinuses, fractures, 277–279
Paraplegia:
  care, 265
  decubitus ulcers in, 264
  laminectomy in, 260
Paresis, of limbs, 53
Paresthesia, in cold injury, 41
Patching, in treatment of eye injuries, 284, 286–287
Patella, fracture, 231
Pathogens, sensitivity to antibiotics, 126–127
Patients:
  litter, transportation, 181
  movement, 159–160, 177
  See also Evacuation; Mobility.
Pelvic abscesses, 336–337
Pelvic area, wounds associated with hip joint injury, 232
Pelvis, fractures, 350
Penicillin, 29, 125, 127, 132, 141, 142, 147, 294, 333
Penicillin, procaine, 132
Penicillin G, 132, 140, 144, 150
Penis, wounds, 350
Penrose drain, 275, 278, 279, 325, 330
Pericardial tamponade, 309
Pericardiocentesis, 308, 309, 311
Pericranium, 250, 251

Perineum:
burns, 30
infection of wounds, 206
Peripheral nerves:
causalgia, 236
classification of injury, 234–235
closed injuries, 236
splinting, 236
wound care, 235–236
wounds and injuries, 183, 234–237
See also Nerves.
Peritoneum, wound closure, 202
Peritonitis, 53, 54
as sequela in abdominal wounds, 315
infection as cause of death, 123
in overlooked abdominal wound, 343
postoperative, 335–338
with fistulas, 346
Peroneal nerve, injuries, 236
Petechiae, subcutaneous, 78
Pharynx:
edema, 167
infection, 269
wounds, 297–298
Phenobarbital, 149
pHisoHex, 44, 45
Phlebitis, 103
Phlebotomy, 101, 334
Phosgene, 59–60
Phosphorus burns, 19, 23, 31–32, 285–286
Phosphorus pentoxide, pulmonary irritant, 32
Physical therapy, in spinal cord injuries, 265–266
Physiology, responses to trauma, 104–118
Physiotherapy, for causalgia treatment, 237
Physostigmine salicylate, as antidote for glycolates, 60
Pigment:
deposition in kidneys, 203
in urine, 204
leakage in crush injury, 203
Pitressin, 343
Plasma. *See* Blood plasma.
Plasma expanders, after trauma, 105
Plasmanate, 25, 102
Plaster casts. *See* Casts, plaster.
Plastic repair, in maxillofacial wounds, 267
Pleural effusion, 337
Pleural space, 313
evacuation, 312, 313
Plutonium, 71
Pneumomediastinum, tracheostomy complicated by, 302
Pneumonitis, 52, 60
Pneumoperitoneum, 53, 54
Pneumothorax, 175
open, 304
pathophysiology, 304
tension, 158, 176, 304, 309, 310
tracheostomy complicated by, 302
Polymyxin, 127, 133
Polymyxin B, 126, 286
Pontocaine, 284
Popliteal artery, 218, 224
Popliteal vessels, 220
Position, of soldier at wounding, 16, 282
Position of function—
for joint dislocation, 233
for splinting hand, 230
of hand, 184, 236, 353, 354, 357
Postoperative management, 195, 220–222
Postoperative management, in—
abdominal wounds, 332–335
amputation, 243
chest wounds, 314
hand wounds, 357
head injuries, 251
maxillofacial wounds, 273
soft-tissue wounds, 206–207
spinal cord injuries, 262–266
Potassium:
antagonism by calcium, 113
excretion, 106
intoxication in renal failure, 110, 111
leakage in crush injury, 203
reincorporation, 113
release into blood, 112, 205
replacement therapy, 334–335
retention in blood, 204
serum, 25, 334
See also Hyperkalemia.
Precession, 14
Preoperative preparation, 188–189, 197–198, 354
Pressure:
for hemorrhage control, 177, 211
intracranial, 251, 254
intrapleural, 176
pleural and pericardial, 307
Pressure sores. *See* Decubitus ulcers.
Pressure waves, 48, 49

Pressurization, of aircraft, 162
Priorities, 158
See also Sorting.
Priorities, in—
abdominal injury surgery, 316–317
arteriovenous fistulas, 213
burns, 18
chemical injury, 56–57
chest wounds, 310
crush injuries, 69
head wounds, 249–250, 256
multiple injuries, 83, 84, 85
nuclear warfare, 63–64
soft-tissue wounds, 198
surgery, 157
thoracoabdominal wounds, 310
vascular injury, 208, 209
Probenecid, 287
Procaine hydrochloride, 223
Program, for cold injury prevention and control, 46
Proparacaine hydrochloride, 282, 284
Prostate, injury, 350
Prosthesis:
application in amputation, 243
in arterial repair, 217
in maxillofacial wounds, 267
Marlex mesh, 345
Prostigmin, 263
Protective mask, 56, 58, 59, 60–61, 172
Protective measures and equipment, 16, 19
Protein:
deficiency, in trauma, 107
depletion, 264
dietary, 259
Proteinuria, 109
Proteus, 292
antibiotic sensitivity, 127
in war wounds, 122
Prothrombin time, 118
Pseudomonas, 286, 287, 292
antibiotic therapy, 133
in war wounds, 122
Psychological factors, in cold injury, 40
Psychological symptoms, in causalgia, 237
Pulmonary insufficiency. *See* Respiratory insufficiency.
Pulmonary shunting, 116
Pulse:
after arterial repair, 216–217
Pulse—Continued
after trauma, 105
neurological import, 249
Pulse, in—
abdominal injury, 334
arterial patency, 212
blast injury, 51
cardiac tamponade, 307
clostridial myositis, 137
hypovolemia, 104
postoperative peritonitis, 336
shock, 91, 93, 94
vascular injury, 210
Pupils:
constriction, 57
dilatation, 254
irregularities, 283
response, neurological import, 248
Pus, release, 233
Pyelography, 316, 348
Pyloroplasty, 117, 343
Pyrexia. *See* Body temperature; Fever.
Quadriplegia, from spinal cord injury, 300
Races:
cold injury protection, 47
susceptibility to cold injury, 40
Radial artery, 214, 354
Radiation:
dose, response, 79–81
dosimetry, 81
from nuclear detonation, 62
particulate, 69
prompt, 70, 73, 80
residual, 70
See also Beta radiation; Gamma radiation; Irradiation; Neutron radiation; Thermonuclear warfare.
Radiation injuries, 62, 64, 65, 66, 68, 69
as component of multiple injuries, 82
mass casualties, 69–81
Radiation sickness, 69, 74–79
Radioactive fallout, 70, 72
decay, 70, 71
decontamination, 65
formation, 72–73
Radiography. *See* Roentgenographic examination.
Radiopaque iodine componds, 344
Rales, 51
Rash, with Sulfamylon use, 33
Recording, 180–181
before anesthesia and surgery, 187
by anesthetists, 187

Recording—Continued
of fracture data, 228
of narcotic administration, 180
Recording, in—
bone and joint injuries, 226
burn injuries, 34
head wounds, 249, 256
nerve injuries, 234, 235
spinal cord injuries, 258
Rectum:
blast injury, 54
wounds and injuries, 326, 329–331
Reflexes, neurological import, 249
Renal damage, pigment deposition as factor in, 203
Renal dialysis, in crush injury, 206
Renal dysfunction, in crush injury, 204
Renal failure, 207, 338
after revascularization, 223
in electric injury, 26
oliguric, 335
Renal insufficiency:
acute, 109–114, 203, 220
See also Kidney; Uremia.
Reoperative abdominal surgery, 340–346
Resection, of—
colon, 326–329
small intestine, 325
Respiration:
accidents stopping, 167
artificial, 167–174
in shock, 93, 94
neurological import, 249
paradoxical, 176, 307
positive pressure, 312
See also Mouth-to-mouth artificial respiration; Mouth-to-nose artificial respiration.
Respirator:
in air evacuation, 160
volume-cycled, 314
Respiratory arrest:
emergency care, 166
in tetanus, 145, 150
Respiratory changes, in blast injury, 51
Respiratory complications:
due to nerve agents, 57
prevention, 253
Respiratory deficiency syndrome, 332–333
Respiratory distress, 52, 60, 145
Respiratory embarrassment, in spinal cord injury, 263
Respiratory insufficiency, 27–28, 114–116, 338
Respiratory irritation, 61
Respiratory obstruction:
emergency care, 166, 167
in maxillofacial wounds, 269, 279
priority of treatment, 158
Respiratory restriction, escharotomy for, 29
Respiratory tract:
action of irritants on, 58
burns, 83
nerve agent absorption, 57
sensitivity to vesicants, 58
Resuscitation, 91–103
facility organization, 95–97
in burns, 18
in multiple injuries, 84
See also First aid.
Retroperitoneum:
hemorrhage in, 344
injuries, 343
Rewarming, for cold injured parts, 43, 44
Rhinorrhea, 268, 275, 278
Rib, fracture, 51, 176, 307
Rifle, bullets, 10, 11
Rifleman, cold injury in, 39
Ringer's lactate solution, 24, 101, 107, 178, 179, 180, 334
Risus sardonicus, in tetanus, 145
Robinson catheter, 350
Roentgenographic examination:
before air evacuation, 161
facility for, 95–96
for abscesses, 337
of abdomen, 344
of chest, 304, 309, 310, 311, 313, 314
Roentgenographic examination, in—
abdominal wounds and injuries, 316, 333–334, 343
blast injury of chest, 52
cervical spine injuries, 262
fractures, 227
head wounds and injuries, 249, 250, 251, 254
joint injuries, 231
larynx and trachea wounds, 296
maxillofacial wounds, 268
multiple injuries, 85
pharynx and esophagus wounds, 297–298
soft-tissue wounds, 197

Roentgenographic examination, in—Continued
spinal cord injuries, 258
sprains and dislocations, 233
Roux-en-Y choledochojejunostomy, 324
Roux-en-Y drainage, 323
Rule of nines, 19
Sacrum, pressure sores, 229
Safety precautions, against burns, 19
Saline solution:
isotonic, 24, 114
normal, 101, 180, 334
Salivation, 58
Salt, deficiency in trauma, 106, 108
Saphenous vein, 217
Scalp, debridement, 250
Sciatic nerve, 236, 240
Sclera, laceration, 283, 289–290
Scopolamine hydrobromide, 284
Scrotum, wounds, 350
Secretions, evacuation, 187, 195
Sedation, contraindication, 253, 259
Sedation, in —
blast injuries of abdomen, 54
peritonitis, 338
tetanus, 149–150
Sensory disturbance, 258
Sepsis, 86, 111
abdominal, 331, 338
soft-tissue, 199
stress ulcer hemorrhage in, 343
Septicemia, 338
Septic shock, 92, 94–95, 142
Sequestration, in maxillofacial wounds, 267
Serosal patching, 325
Serotherapy, in—
clostridial myositis, 142
tetanus, 148
Serratia marcescens, 17
infection, 333
in war wounds, 122
Serum. *See* Blood serum.
Service, branch, role in cold injury, 39
Shock, 91–103, 111
antibiotic therapy, 132
clinical manifestations, 93–95
evaluation in multiple injuries, 85
muscle relaxants in, 194
neurological import, 249
pathogenesis and etiology, 91–92
postoperative control, 195
predisposing factors, 92
priority of treatment, 158
treatment, 97–103, 178–179
Shock—Continued
triage and resuscitation facility, 95–97
See also Neurogenic shock; Oligemic shock; Septic shock.
Shock, in—
abdominal injury, 316
amputation, 184
blast injury, 48, 50, 51, 53
crush injury, 203, 204, 205
maxillofacial wounds, 269
multiple injuries, 83
Shoulder joint, splinting, 230
Silver nitrate, wet soak therapy, 34
Silvester method. *See* Chest pressure-armlift artificial respiration.
Sinuses:
dural, 255
paranasal, fractures, 277–279
venous, 255
Sinusitis, 277
Skeletal traction, 219
Skin:
breakdown, 228, 253
changes in clostridial myositis, 138–139
characteristics in cold injury, 41
characteristics in nerve injury, 234
color, 59, 61, 93, 94, 137
damage, amputation in, 239
elasticity loss, 108
electrolyte loss, 106
nerve agent absorption, 57
pressure necrosis, 236
sensitivity to vesicants, 58
traction, 161
water loss from, 107
Skin, in—
halothane anesthesia, 193
hypovolemia, 104
shock, 91
Skin flaps, 271
in amputation, 240, 242, 243
in scalp wounds, 251
Skin grafts, 46, 232, 243, 251, 345
Skull, debridement, 250
Small intestine, wounds, 325
Smoke inhalation, 27
See also Inhalation injury.
Smokes, dangerous, 61
Smoking, contraindication, 45
Sodium:
cautious use, 114, 115
conservation, hormonal regulation, 106

Sodium—Continued
excretion, 106
level in renal failure, 110
level in serum, 107
loss in trauma, 108
Sodium bicarbonate, 102, 114
Sodium chloride, level in plasma, 106
Sodium nitrite, 59
Sodium thiosulfate, 59
Soft-tissue wounds, 196–207
crush injury, 203–206
debridement, 198–202
management, 196–197, 206
postoperative management and evacuation, 206–207
preoperative preparation, 197–198
priority of treatment, 158
Solid blast, 49
Sorbitol, 113
Sorting, 153–158, 159
at battalion aid station, 156
at initial wound surgery level, 157
concepts, 157
facility, 95–97
in nuclear warfare, 63–64
See also Priorities.
Sorting, in—
abdominal injuries, 316–317
burns, 66
cold injuries, 47
multiple injuries, 83–84
radiation injuries, 81
Spasm:
arterial, 210
muscle, in vascular injury, 210
segmental occlusive, 223
vascular, 209, 222
Spermatic cord, wounds, 350
Sphenoidal bone, 268
Sphenoidotomy, 279
Sphenoid sinus, wounds, 279
Sphincter, urethral, 109
Spinal cord injuries, 93, 158, 182, 257–266
classification, 257
closed, 259–260
evaluation, 258
from neck wounds, 300
management principles, 259
open, 260
physical therapy, 265–266
supportive and postoperative care, 262–266
Spine, arching, 145
Spleen, injury, 322
Splenectomy, 322
Splinting:
regional, 230–231
See also Immobilization.
Splinting, of—
crush injuries, 205
hand, 184, 353, 357
mandibular fractures, 274
palate, 277
peripheral nerve injuries, 236
urethral injuries, 349
Splints, 179
cock-up, 236
for amputation, 185
wire, for jaw, 271–272
wire ladder, in amputation, 243
Sprains, 233
Sputum, culture and smear, 333
Staphylococci:
antibiotic-resistant, 127
antibiotic therapy, 133
hemolytic, antibiotic sensitivity, 127
hemolytic coagulase-positive, 122
in clostridial myositis, 137
in otitis, 292
Staphylococcus aureus, 122, 124
Steinmann's pins, 228
Stenosis:
prevention, 214
tracheal and laryngeal, 302
Sternotomy, 312
Sternum, fracture, 307
Steroids, 253
contraindication, 285
in smoke inhalation, 28
Stomach:
contents, aspiration, 188
evacuation, 84, 188, 319
pathologic response to trauma, 116–117
wounds, 324
Stool. *See* Feces.
Streptococcal myositis, 139, 144
Streptococci:
anaerobic, antibiotic sensitivity, 127
beta-hemolytic, 29, 122, 127
hemolytic, 124
in clostridial myositis, 137
Streptomycin, 127
Stress, 81
Stress ulceration, 116–117, 338, 343
Stryker frame, 162, 260, 262
Subarachnoid space, 261

Subcutaneous tissue, for covering arterial repair, 218
Subphrenic abscesses, 336–337
Succinylcholine, 194
Sucking chest wounds, 158, 175–176, 202, 304, 310
Suction, high-volume, 346
Sulfamylon, 33–34, 126, 292
Sulfonamides, 127
Sump drains, 319, 323, 325, 330, 345
Supplies, triage and resuscitation facility, 96–97
Suppuration:
  identification, 123, 124
  local, 124–125
Surgery:
  conditions, of soft-tissue wounds, 197
  early and adequate, 119, 120, 140, 141, 147, 197, 210
  for anaerobic cellulitis, 143
  for streptococcal myositis, 144
  forward, 1–5
  military, principles, 154
  policy in multiple injuries, 86
  priorities for, 157, 208
  reoperative, abdominal, 340–346
  See also Debridement; Excision; Incision; specific types of surgical procedures.
Surgical meetings, 4
Surgicel, 216
Survival rate:
  after vascular injury, 208
  increase, 155
  See also Mortality.
Suture repair, of—
  arterial laceration, 212, 214
  hepatic tissue, 322
Sutures, retention, 331, 341
Suture technique:
  anastomosis, 214–216
  in corneal and scleral wounds, 289–290
  in maxillofacial wounds, 270–271
  of eyelid wounds, 288
Sweating, loss in nerve injury, 234
Swelling, 276, 277
  elevation to control, 228
  muscular, after vascular injury, 223
  postoperative, 219
  See also Edema.
Swelling, in—
  clostridial myositis, 138
Swelling, in—Continued
  crush injury, 203, 204, 205
  maxillofacial wounds, 268, 269
Sympathectomy:
  chemical, 191
  contraindication, 222
  for causalgia treatment, 237
Sympathetic block—
  for causalgia treatment, 237
  in vascular injury, 222
Syncope, in shock, 92, 93
Syndromes:
  central nervous system, 74, 75–77
  gastrointestinal, 74, 75, 77–78
  hematopoietic, 74, 75, 78
  respiratory deficiency, 332–333
  "wet lung," 314
Synovium, 231–232
  wound closure, 202
Tachycardia, in trauma, 94, 104
Tachypnea, 332
Teamwork, in—
  maxillofacial care, 267
  military medicine, 1
  multiple system wound care, 270
Tear and nose agents, 58
Teeth:
  alinement, 268, 273
  management, in facial wounds, 271
  See also Malocclusion.
Temperature:
  body. *See* Body temperature; Fever; Hypothermia.
  role in cold injury, 37
Temporalis fascia, 251
Temporomandibular joint function, 268
Tendon:
  management in debridement, 200
  repair, 227, 355
Tension:
  intraocular, estimation, 283
  relief in soft-tissue wounds, 206
Testes, wounds, 350, 351
Tetanus, 144–150
  as cause of death, 123
  clinical picture and diagnosis, 145
  differential diagnosis, 145
  management, 148–150
  prophylaxis, 29, 146–148, 198, 286, 292
Tetanus toxoid, 44, 179
  See also Toxoid, tetanus.
Tetracaine, 191, 284

Tetracyclines, 125, 127, 132, 140, 141, 142, 144, 147, 333
Thermal injury:
  of external ear, 292
  See also Burns; Cold injury.
Thermonuclear devices, burns from, 18
Thermonuclear explosion, burns from, 21
Thermonuclear warfare:
  mass casualties, 62–81
  medical principle in, 4–5
  See also Nuclear weapons.
Thigh:
  anastomosis in, 216
  burns, 30
  injury, tetanus in, 145
  splinting, 179
Thiopental, 141, 149, 193
Thirst, 112
Thoracoabdominal wounds, 86, 309–310, 313
Thoracostomy, closed tube, 308–309, 311
Thoracotomy, 298, 311–313
Thorax:
  arterial wounds, 217
  vascular injury in, 208
  wounds, 158, 249
  See also Chest wounds.
Thorazine, 92
Thrombophlebitis, septic, 140
Thromboplastin time, 118
Thromboplastin time, 118
Thrombosis, 209
  after arterial grafts, 217
  after arterial repair, 223
  from missile cavitation, 209
  of vein graft, 218
Thrombus, 216
  arteriographic identification, 222
  clearing, 212
  formation in arterial wounds, 212–213
Thumb, amputation, 355
Tibia, fracture, 120
Tibial artery, 214
Tinnitus, 294
Tissue, destruction, 13–14
Tissue perfusion:
  after trauma, 104, 105
  hormonal regulation, 105
  improvement, 193, 194
  in shock, 92
Toes:
  clawing, 266
  management in splinting, 231
Tongue
  characteristics in salt depletion, 108
  loss of control, 167
  prolapse, 269
  swelling, 269
Torticollis, 258
Tourniquets, 158, 243
  amputation after, 211
  for hemorrhage control, 177–178
  pneumatic, 211–212
  prolonged application, 136
  rotating, 101
Tourniquets, in—
  amputation, 240, 243
  bone and joint injuries, 226, 227
  extremity injury, 184
  hand surgery, 354
  soft-tissue wounds, 197, 198
Toxemia, in clostridial myositis, 138
Toxicity, systemic, 92
Toxin:
  bacterial, in war wounds, 122
  in septic shock, 94
  tetanus, 144, 145, 148
Toxoid:
  tetanus, 146, 147
  See also Tetanus toxoid.
Trachea:
  displacement, 304
  wounds, 296–297
Tracheobronchial inflammation, 32
Tracheobronchial secretions, 314, 333
Tracheobronchial tree, noxious drying, 115
Tracheobronchitis, from inhalation injury, 27
Tracheostomy, 114, 115
  care in air evacuation, 160–161
  emergency, 297, 300–302
  water loss with, 107
Tracheostomy, in—
  blast injury of chest, 52
  burns, 27–28
  chest wounds, 308, 309
  head wounds, 249
  maxillofacial injuries, 183, 279–280
  respiratory emergency, 167
  tetanus, 150
Traction:
  effect on anastomosis, 219
  elastic, for jaw, 271, 274, 277
  of cranial tongs, 161
  skeletal, 228
  skin, 161

Traction, in—
amputation, 242, 243–244
cervical spine injuries, 262
fractures associated with burns, 80
Transection, of—
blood vessels, 209
spinal cord, 257, 259
Trauma:
hyperkalemia in, 194
physiologic responses to, 104–118
Trenchfoot, 35, 36, 37, 39
Trephination, 254
Triage. *See* First aid; Sorting.
Trismus, in tetanus, 145
Trunk:
circumferential burns, 28
protection from blast injury, 54
T-tube drainage, 320, 324, 348
Tumbling, 14
Tympanic membrane:
injuries, 48, 49, 50–51, 293
protection, 54
Ulcers, stress, 116–117, 338, 343
Ulnar artery, 214, 354
Ultraviolet burns, of eye, 285
Underwater (immersion) blast, 49
United States. *See* Zone of Interior.
Uranium, 71
Uremia, 110, 111, 204
See also Kidney; Renal insufficiency.
Ureter, 109, 348
Ureterostomy, 348
Urethra, 109, 349–350
Urethral stricture, 330
Urethrography, 349
Urinalysis, 316, 348
Urinary drainage, 251, 264
See also Drainage.
Urinary output, 102, 188
after arterial surgery, 220
reduced, 106
Urinary output, in—
abdominal injury, 334
abdominal surgery, 334
air evacuation, 162
burns, 26–27
clostridial myositis, 142
crush injury, 205
shock, 94
spinal cord injuries, 264
trauma, 108–114
Urine:
characteristics in crush injury, 204
Urine—Continued
characteristics in renal failure, 109, 111
electrolyte loss, 106
Vagotomy, 117, 343
Valsalva maneuver, 294
Valves, vascular, 217
Vascular clamp, 211, 214
Vascular injuries, 158, 208–224
associated injuries, 219–220
care in air evacuation, 162
complications, 222–223
conservative management, 213
debridement, 212–213
diagnosis, 209–210
gangrene from, 239
hemorrhage control, 177, 211–212
nonoperative treatment, 213
of neck, 298–300
postoperative management, 220–222
principles of care, 209
priority over head wounds, 249
repair, 214–219, 224, 226, 227, 238
surgical timing, 219
See also Blood vessels.
Vascular system, peripheral, 192
Vasoconstriction:
inhibition, in shock, 93
in shock, 91, 100
in trauma, 104
Vasodilatation:
cold induced, 37, 40
in shock, 93
Vaso-pressors, 103, 106, 187
Vein grafts, 214, 217, 218,
Veins:
concomitant, management, 217–218
for catheter insertion, 98
for intravenous administration, 178
injuries, 220
See also Blood vessels; Vascular injuries.
Velocity, of missiles, 10–13
Vena cava, intrahepatic, hemorrhage, 320
Venous sinuses, 255
Ventilation:
assisted, 28, 56, 57, 60, 61, 86, 116, 188, 209
improvement in chest wounds, 310
maintenance in chest wounds, 314
minute, 307–308
positive pressure, 309
Ventilator, volume-controlled, 333

Vertigo, 294
Vesicants, 55, 58
Vietnam war:
  amputation rate and arterial repair, 224
  care of wounded, 4
  clostridial myositis in, 140
  distribution of missile injuries, 16
  evacuation in, 155, 156
  neurosurgical care in, 247
  rifles, 11
  Serratia infection during, 333
  sorting in, 154
Vinke tongs, 262
Viscera:
  blast injury, 48
  displacement, 337
  hollow, wounds, 324–331
  injuries, 158
  solid, wounds, 319–324
Visual acuity, evaluation, 282–283
Volkmann's ischemic contracture, 223
Vomiting, 74, 93
  in clostridial myositis, 137
  salt depletion with, 108
Vomitus, aspiration, 167
Warm packs, 223
War Surgery Conferences, 4
Water:
  conservation, hormonal regulation, 106
  depletion, in trauma, 107
  physiologic response to trauma, 106–108
Wavelength, in airblast, 48
Weakness, muscular, 60
Weather, role in cold injury, 38
"Wet lung" syndrome, 314
Whirlpool bath, for cold injury, 44, 45
Wind, role in cold injury, 38
World War I:
  hydrogen cyanide use in, 59
  rifles, 11
World War II:
  clostridial myositis in, 140
  cold injury in, 35, 39, 40
World War II—Continued
  distribution of missile wounds, 16
  sorting, 154
Wound ballistics:
  definition, 9
  fundamentals, 9–15
Wound closure:
  abdominal, 331
  delayed, 196–197, 207, 220, 225, 227, 232
  primary, 270–271, 289
  stump, 243
  timing and method, 242
Wound closure, in—
  chest wounds, 313
  eye wounds, 289
  hand wounds, 356–357
  head wounds, 251
  maxillofacial wounds, 267
  pharynx and esophagus wounds, 298
Wounds:
  characteristics favorable for clostridial myositis, 136
  dehiscence, 331, 341
  missile-caused. *See* Bullet wounds; Missile-caused wounds.
  repair process, 196
  sepsis, 199
  types, 13
Wound suppuration:
  identification, 123, 124
  local, 124–125
Wound track, 11, 14, 16
  bacterial infection, 17
  incision for debridement, 199
Wrist, splinting, 230
Wristdrop, splint for, 236
X-ray examination. *See* Roentgenographic examination.
Yawing, 14
Zinc peroxide, 149
Zone of Interior:
  colostomy closure in, 332
  evacuation to, 3
Zygoma, 274, 275, 276

U.S. GOVERNMENT PRINTING OFFICE: 1976 O—201-745